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A TREATISE

ON

THE CONTINUED FEVERS

OF

GREAT BRITAIN.

BY

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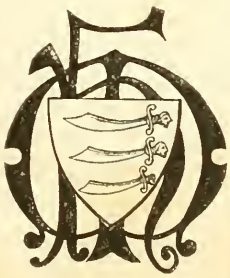

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TO

THOMAS WATSON, M.D. F.R.S.

PRESIDENT OF THE ROYAL COLLEGE OF PHYSICIANS

OF LONDON,

THIS WORK IS RESPECTFULLY DEDICATED,

BY

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PREFACE.

NO Apology is necessary for offering to the profession a Treatise on the Continued Fevers of Great Britain; as no work of the kind has been published by any English physician for nearly a quarter of a century, notwithstanding the great advance of late years in our knowledge of the diseases in question. Some account, however, may be expected of the author of a work on what is acknowledged to be one of the most difficult subjects in Medicine. During my connection with the London Fever Hospital, extending over upwards of six years, I have had unusually favourable opportunities for studying the diseases of which I treat. I was also a clinical clerk in the Edinburgh Royal Infirmary, during the great epidemic of typhus and relapsing fever in 1847-8. Afterwards, I studied fever for several months in Dublin and Paris; and, while serving with the army in India and Burmah, I had the advantage of being able to compare the fevers of tropical climates with those of this country. Lastly, having twice suffered from one of the diseases which I have here attempted to describe, I may adopt the plea, by which Thucydides justified himself, in writing the history of the Plague of Athens: '*ταύτα δηλώσω αὐτός τε νοσήσας, καὶ αὐτὸς ἰδὼν ἄλλους πάσχοντας.*'

It has been my humble endeavour, in this work, to follow the example of Louis, and, wherever it has been practicable, to reduce my observations to a numerical expression. Some writers object to the application of statistics to medical science, and prefer trusting to what they call experience. But experience, to be of value

to any one besides the immediate observer, must be something capable of definite expression. Moreover, the mind is apt to attach to accidental occurrences an importance, which is at once dispelled by an appeal to the 'force brutale de chiffres.'

A feature unusual in a practical work, is the large share of attention here devoted to the Causes of Fevers. My conviction that Continued Fevers are diseases which may be prevented, and the circumstance that the questions discussed have occupied greatly the attention of scientific men of late years, induce me to think that my remarks on this subject will be of service, if it be only in stimulating other observers to further investigations, for the purpose of testing the correctness of the conclusions at which I have arrived. The history of Continued Fevers, possessing, as it does, an importance which does not attach to the history of most other maladies, has also been considered at some length: it involves an account of some of the greatest calamities which have befallen our race, and it teaches important lessons, by means of which we may hope to prevent similar calamities in future.

In discussing certain topics, I have not hesitated to express freely my own opinions, although they are occasionally at variance with those of some of my professional brethren, for whose judgment I entertain profound respect. But, where this has been the case, I have adduced the evidence on which I have based my dissent, and I trust that I have not been wanting in that deference to the opinions of others, which ought to characterize all scientific discussions. With regard to the specific distinctness of typhus and enteric fever, it is right to state, that I was taught to regard them as mere varieties of one disease; and that, with this impression, I commenced their study at the London Fever Hospital. If my subsequent observations, aided by the convincing arguments of Drs. Stewart and Jenner, have led me to an opposite conclusion, it cannot be said that my present convictions are the result of preconceived opinions. Whatever be the decision arrived at on this subject

and on other disputed points, many of the observations collected in this volume have an important bearing on the questions at issue, and 'I know that the truth is in the facts, and not in the mind 'which observes them.'

In the treatment of each subject, I have given the results obtained by other observers, as well as by myself, and I have collected, in a Bibliography, the more important monographs and essays referred to in the text. The references throughout are restricted to the author's name, with the date and page of the work. The full title of the work will be found by referring to the Bibliography. By adopting this plan, much needless repetition has been avoided, while, at the same time, an attempt has been made to bring together the more important works on Continued Fevers, and thus to supply a want which has been often complained of.

In addition to the forty-four illustrative cases selected from many hundreds, of which I have notes, I have given throughout the work, the results of an analysis of numerous cases reported by myself on a uniform plan, the notes being taken daily on printed sheets, with a heading for each symptom. Many of the statistical tables referring to the etiology and mortality of Continued Fevers were contained in an essay which I read to the Royal Medical and Chirurgical Society of London in 1858. These tables were compiled with great labour and care from the Registers of the London Fever Hospital, extending over a period of ten years; and most of them have now been brought down to the present date. It is believed that the statistics of a hospital, where the different fevers have been distinguished for nearly fifteen years, cannot fail to be useful.

Many of the Tables contained in the essay referred to, together with my remarks upon them, have been adopted by Dr. TWEEDIE, in his Lumleian Lectures, published in the '*Lancet*' for 1860. Dr. TWEEDIE being about to republish his Lectures, I feel it due

to myself further to state, that most of his facts and reasonings, bearing on the question of the 'change of type' of Continued Fevers, will be found in a paper published by me in the '*Edinburgh* '*Medical Journal*' for August, 1858. As Dr. TWEEDIE omits to mention my paper, I think it necessary to allude to the circumstance, lest it might appear that I had now borrowed some of my remarks from his Lectures, without acknowledgment.

The coloured plates of the cutaneous eruptions met with in Continued Fevers, have been successfully drawn from nature by Dr. Westmacott, and copied on stone by Mr. William West under my superintendence, and they will help to make the descriptions given in the text more intelligible.

79, WIMPOLE STREET, CAVENDISH SQUARE.
October 15th, 1862.

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CORRIGENDA AND ADDENDA.

Page 32, line 39, *omit* portion of sentence after 'poor,' which is erroneously quoted.

„ 37, *For* "967 cases admitted into the London Fever Hospital in 1848," *read* "1042 cases."

„ 54, line 3. Since this paragraph was written, Typhus Fever has broken out at Preston. On October 5th, Mr. R. C. Brown, House Surgeon of the Dispensary, wrote to me that during the preceding ten days, 25 cases of a very fatal form of typhus, 'with a well-marked mulberry rash, had been admitted into the Fever Hospital attached to the Preston Union. Since that date, the number of cases has greatly increased. As far as I have been able to ascertain, this outbreak, which I have long anticipated, has been due to overcrowding consequent on destitution. (See pages 78, 113.)

„ 60, line 10, *for* 'Section IV.' *read* 'Section V.'

„ 217. In reference to Table X., the reader is referred to note at page 604.

A TREATISE

ON

CONTINUED FEVERS.

CHAPTER I.

INTRODUCTION.

CORRIGENDUM.

Page 471, in the Table; No. 8, in the Column headed *Pythogenic Fever*, *for*, Appear on 4th or 5th day, *read*, Rarely appear before 7th day; and No. 8, in the Column headed *Typhus*, *for*, Rarely before 7th day, *read*, Appear on 4th or 5th day.

The voluminous literature on the subject of Fevers, proves the interest attached to them by medical men, in all ages, down to the present day. Like other contagious and epidemic diseases, which are due to the action of a specific poison, Continued Fevers possess a peculiar attraction for the medical philosopher, inasmuch as their study involves an investigation, not merely of their symptoms, pathology, and treatment, but more especially of the causes of their varying prevalence at different periods, and of the laws that regulate their origin and propagation; while, at the same time, a knowledge of fever in the abstract, is indispensable for the study and treatment of all acute diseases. 'In the whole range of 'human maladies,' said Graves, one of the greatest authorities on the subject, 'there is no disease of such surpassing interest and 'importance as fever.'

But the advantages derived from a study of Continued Fevers

A TREATISE

ON

CONTINUED FEVERS.

CHAPTER I.

INTRODUCTION.

THERE are few medical subjects of such interest and importance to the general public as Continued Fevers—a circumstance at once accounted for by their extensive prevalence. From the Registrar General's Reports, it appears, that during the last twenty years they have destroyed 350,000 of the population of England and Wales, and that the average annual mortality from them in London has been upwards of 2,000. The actual number of persons attacked, represented by this mortality, has probably amounted to 3,500,000 in England and Wales during the last twenty years, and reaches 20,000 annually in London alone. At the present moment, Continued Fevers are attracting an unusual share of public attention, from their having blighted the fondest hopes of more than one nation in Europe.

The voluminous literature on the subject of Fevers, proves the interest attached to them by medical men, in all ages, down to the present day. Like other contagious and epidemic diseases, which are due to the action of a specific poison, Continued Fevers possess a peculiar attraction for the medical philosopher, inasmuch as their study involves an investigation, not merely of their symptoms, pathology, and treatment, but more especially of the causes of their varying prevalence at different periods, and of the laws that regulate their origin and propagation; while, at the same time, a knowledge of fever in the abstract, is indispensable for the study and treatment of all acute diseases. 'In the whole range of 'human maladies,' said Graves, one of the greatest authorities on the subject, 'there is no disease of such surpassing interest and 'importance as fever.'

But the advantages derived from a study of Continued Fevers

are not limited to the medical profession. Depending as they do on causes, which, to a great extent are under human control, their study is of special import to the military commander, to whom a healthy army is one of the most essential elements of victory; to the medical jurist, who ought to know, that limited outbreaks of fever have often been attributed to criminal poisoning; to the statesman engaged in framing laws for the health of the people; to the sanitary reformer, and to the community at large, whose duty and interest it is to avert disease and death.

Continued Fevers have been classed by medical writers of all ages as distinct, on the one hand, from the Eruptive, and, on the other, from the Intermittent and Remittent Fevers. But, although this classification may be in some respects convenient, the distinction is on both sides entirely arbitrary. Some of the continued fevers agree with the eruptive in being eminently contagious, in rarely attacking an individual more than once, and in being characterised by the presence of a peculiar eruption on the skin; while, on the other hand, one of them (simple fever) is not at all contagious; another (pythogenic), is but slightly so; in two (relapsing fever and simple fever), one attack confers no immunity from subsequent attacks; in two (relapsing fever and simple fever) there is no specific eruption; one of them (pythogenic) frequently assumes a remittent type, so as to resemble remittent fever; and all of them are thought to agree with the malarious fevers, but to differ from the eruptive, in arising from preventible causes, or in being capable of spontaneous generation. Hence the diseases known as 'Continued Fevers,' constitute a somewhat heterogeneous class, and may be said to occupy an intermediate position, between the eruptive and malarious fevers.

Many of the early writers on medicines, such as Riverius, Willis, Hoffmann, Strether, Huxham, Pringle, and Macbride, recognised and described different forms of Continued Fever; but their investigations did not suffice to establish absolutely the specific non-identity of the diseases which they observed. During the last twenty years, no subject has occupied more the attention of the profession, or created greater discussion, than that of the specific identity or non-identity of the different forms of continued fever. But now the question may be regarded as finally settled. The investigations of Henderson and other writers on the epidemic of 1843, established the specific distinctness of relapsing fever from typhus, while those of Gerhard, Stewart, Jenner, and others have proved the non-identity of the true typhus and the 'typhoid

'fever,' so ably described by Louis. These three diseases, then, are all included under the generic term Continued Fevers, as likewise a fourth, which may be styled Simple Fever. The three former owe their origin to poisons which are probably as distinct as those of Measles, Scarlet Fever, and Small Pox; Simple Fever, arises from non-specific causes, such as exposure to heat, nervous exhaustion, etc. Another circumstance worthy of notice is, that of the three specific fevers, two (typhus and relapsing, but particularly the latter) prevail, for the most part, as great epidemics, whereas the third (pythogenic) has more of an endemic character.

According to our present knowledge, the continued fevers of Britain may be classified as follows:—

- A.—Non-Specific . I. Simple Fever, caused by . . { Exposure to sun, fatigue, surfeit, etc.
- B.—Specific.
  - II. Endemic (Pythogenic, Enteric, or Typhoid) . . . . . { Poison contained in emanations from sewers, &c.
  - III. & IV. Epidemic
    - Typhus caused by . . . { The concentrated exhalations from squalid human beings.
    - Relapsing Fever . Famine.

The plurality of Continued Fevers is now generally admitted, and is advocated in this work. It is true, that there are still some distinguished members of the profession, who believe that the fevers above mentioned are mere varieties, and all spring from one poison. But the opinions of great authorities must not be allowed to bias the mind and make it misinterpret the facts of nature. It must not be forgotten that, among our forefathers, were men characterised by genius and powers of observation, equal to those possessed by any living physicians, who regarded variola, measles, and scarlet fever, as all modifications of one disease — different effects of the same poison,—although their own recorded descriptions prove, that the diseases they saw were as different as they are now. It is, in my opinion, difficult to conceive how any person, who gives the evidence, now accumulated in reference to Continued Fevers, a fair consideration, can arrive at any other conclusion than that they are as distinct as small pox, measles, and scarlet fever; or to account for their failure in so doing, otherwise than on the supposition, that, like some modern physicians and sanitary reformers, they regard not only continued fevers, but small-pox, measles, scarlet fever, the plague, remittent and

intermittent fevers, as all modifications of the same affection, the poison of all being the same.[a] But granting that the different continued fevers were essentially and fundamentally alike, it would still be hardly less important to be able to distinguish them as forms or varieties of disease. In a practical point of view, the necessity of an accurate diagnosis is the same, whether we regard them as species or varieties.

The evidence in favour of non-identity, and the arguments urged in support of identity, will engage our attention hereafter: but, in the mean time, it may be well to mention some of the circumstances which, for so long a period, led to the different continued fevers being confounded, and which have not ceased to operate at the present time. They are mainly the following:—

1. Observers, who have had experience in only one form of Continued Fever, have naturally thought that all cases resembled those which came under their own notice, and have consequently arrived at the conclusion that there is but one species. It is thus that many distinguished physicians in France, whose experience was limited for the most part to the so-called '*Fièvre typhoide*,' found it difficult to believe in the existence of typhus, as a distinct affection; while, on the other hand, the comparatively few cases of the French fever observed at Edinburgh were regarded as a complicated variety of the true typhus, which was there so prevalent.

2. Arguments have been frequently based on the name assigned to a disease prevalent at a given time or place, instead of on the disease itself. It is a remarkable fact, that several writers have argued as if previous observers had employed the terms Typhus, Typhoid, etc., with strict accuracy, when they themselves failed to recognise any specific distinction between the diseases in question.

3. Different fevers have frequently been epidemic at the same time, and the published descriptions have included both, as one disease, under one name.

4. In the case of Relapsing Fever, the relapse has often not been recognised, from the patient being seen in one of the attacks only.

5. Much confusion has arisen from the undefined meaning attached to the term *petechiæ*. In its ordinary acceptation, this word implies small circumscribed extravasations of blood in the

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[a] See SMITH, 1830, p. 75; HENDERSON, 1843, p. 202; Miss NIGHTINGALE'S Notes on Nursing, p. 19.

substance of the true skin, such as may occur in the course of any specific fever, or even in the advanced stages of other diseases. But by some writers, both ancient and modern, the term has been used to denote the characteristic eruption of Typhus, which has, in consequence, been frequently designated 'Petechial Fever.' Hence, from the occurrence of ordinary petechiæ in pythogenic fever, it has been argued that this affection must be identical with typhus. This subject will be discussed more at length hereafter.

6. There can be little doubt that the eruptions of typhus and of pythogenic fever have been frequently confounded, and that upon mistakes of this nature erroneous arguments have been based.

7. In distinguishing the different forms of Continued Fever, too much reliance has been placed on their symptoms and pathology, while there has been a want of sufficient investigation of their causes. Continued Fevers have many symptoms in common. There is little difference between the *typhoid state* induced by typhus, and the similar condition induced by pythogenic fever. Indeed, if the eruption be absent or indistinct, it may be difficult, from merely seeing the patient in this condition, and knowing nothing of the previous history, to say whether the case be one of typhus or pythogenic fever. But the same difficulty exists in distinguishing typhus from uræmia dependent on kidney disease, and from many other diseases. Morbid affections universally acknowledged to be totally different, and in most cases easily distinguishable, may, under certain circumstances, have many symptoms in common, so as to render their diagnosis difficult. Patients are constantly admitted into the London Fever Hospital, with medical certificates to the effect that they are labouring under contagious fever, whose real disease is not fever, in the strict sense of the term, but some affection of the kidney, brain, or lung. Again, the same fever may exhibit different features, at different times and under different circumstances. Typhus may be complicated with tympanitis, diarrhœa, or dysentery, and so assimilate itself to pythogenic fever, which, in its turn, may exhibit an unusual tendency to cerebral symptoms (the typhoid state), and even constipation, and thus resemble typhus. Moreover, our knowledge of the fundamental pathology of continued fevers is still far from satisfactory. Many other diseases can be distinguished by physical phenomena during life, or by the lesions found after death; but in idiopathic fevers, with one exception, there are no specific lesions. Still, we are not justified in arguing from such facts in favour of the identity of the different forms of continued fever, any more

than we are in maintaining that, because opium produces narcotism, all other narcotics must contain morphia, or that their active principles are identical. It is generally admitted that most continued fevers result from the operation upon the system of some poison; and the main question to be answered is, whether there be, or be not, an *identity of poisons*. To arrive at any certainty in the matter, it is necessary to study the causes of continued fevers in connection with their symptoms. Now, recent investigations have rendered it probable, that the circumstances under which the several continued fevers are generated, are widely different; that the typhus poison is generated by the protracted concentration of the exhalations from living human bodies; that the poison of relapsing fever makes its appearance in that peculiar condition of the system induced by starvation; while that of 'typhoid fever' is contained in the emanations from certain forms of decomposing organic matter. The co-existence of two species of continued fevers in one epidemic is no greater proof of their identity, than is the co-existence of epidemics of scarlatina and variola a proof that these two diseases are the same.

The recognition of several species of Continued Fever explains many of the discrepant statements of different writers. For example, much difference of opinion has existed as to the contagious properties of Continued Fever, but, on inquiry, it is found, that while few who have had any experience of true typhus, doubt the fact of its being contagious, many, whose observation has been limited to pythogenic fever, have been inclined to question the contagious property of any form of Continued Fever. It is obvious, that if the conclusions formed from the observation of pythogenic fever be applied to typhus, the most direful consequences might ensue. Thus, while cases of pythogenic fever may be distributed with impunity among the patients in a general hospital, grave doubts exist as to the propriety of such an arrangement in the case of typhus. Again, while observers of typhus have contended that an eruption upon the skin is rarely absent in Continued Fever, observers of pythogenic fever, in which the eruption is comparatively inconspicuous and often overlooked, and of relapsing fever, which has no characteristic eruption at all, have not unfrequently maintained that the occurrence of an eruption in Continued Fever is quite exceptional. Thirdly, most erroneous conclusions as to treatment have been arrived at, from confounding the different forms of fever. The advocates of blood-letting at the commencement of the present century, appealed to the diminution in the mortality from fever in support of the efficacy of their treatment,

but the reduced mortality was the result, not of the treatment, but of the substitution of relapsing fever for the much more mortal typhus. Lastly, the statements which have been made in reference to fevers having undergone a change of type or nature, are mainly to be attributed to a non-recognition of the existence of different species, together with changes in the prevailing fashion of treatment. A careful study of the history of epidemics shows, that each of the Continued Fevers, and of the other acute specific diseases, has maintained its identity in all ages and countries. Sydenham's descriptions of measles and small-pox are applicable to the measles and small-pox of the present day. The descriptions of typhus by Fracastorius and Cardanus, of relapsing fever by Rutty, and of pythogenic fever by Baglivi, Huxham, and Manningham, correspond exactly with the clinical history of these diseases now. No new specific disease has appeared among us, and the type of each disease has changed little, if at all. Cases of typhus fever occurring during an epidemic of relapsing fever, require stimulants as much as when typhus is itself epidemic, while cases of relapsing fever occurring in an epidemic of typhus will recover, whether left to themselves, or, in spite of blood-letting, as readily as during epidemics of relapsing fever.

But while it is essential, in distinguishing the different species of Continued Fever, to have a due regard to their causes, it is no less necessary to remember the existence of different species of continued fevers, in studying their causes in a sanitary point of view. The neglect of this precaution has been productive of much error, and has greatly impeded the progress of sanitary science. It will hereafter be shown, that, while, on the one hand, it has been contended that fever results from putrid emanations and is quite independent of destitution, on the other hand, it has been urged, that putrid emanations are perfectly innocuous, and that the great source of fever is destitution, with or without overcrowding. The cause of this discrepancy of opinion has been, that the opposing parties have drawn their conclusions from different diseases.

Among the greatest benefits that medicine has conferred on the human race, is the discovery of the causes of disease, and of the measures by which they may be prevented. Recent researches have thrown much light on the causes of Continued Fevers, and render it probable that the belief that these diseases are necessarily in every instance traceable to contagion is erroneous, and that their prevalence is, to a great extent, under human control. Two hundred years ago, agues and other malarious fevers were among the most common diseases of this country. James I and Oliver

Cromwell both died of ague in London, and the latter of these rulers, speaking of ague, makes use of the following oft-quoted words:—'Matrem pietissimam, fratres, sorores, servos, ancillas, nutrices, conductitias, quotquot erant intra eosdem nobiscum parietes, ac fere omnes ejusdem ac vicinorum pagorum incolas, hoc veneno infectos et decumbentes vidi.'[b] The country surrounding London was in Cromwell's time as marshy as the fens of Lincolnshire now are, so that Cromwell was nicknamed 'The King of the Marshes.' But at the present day, owing to the almost universal drainage and cultivation of the soil, agues have, save in a few isolated districts, almost vanished from this country. Again, it would not be difficult to show, that the Oriental plague, formerly so prevalent at different times in London, but since the great fire of 1666 unknown, is not less contagious now than it was in the days of James II, and that its disappearance is due to an improved construction of our dwellings. It is not unreasonable to hope with confidence, for a like extermination of the whole class of Continued Fevers. Already numberless instances might be adduced of the triumphs of sanitary science in extinguishing them, and it is gratifying to find that increased attention to sanitary arrangements was followed during the four years 1858-61 by a diminution of fever in most of the large towns of the British Empire, unequalled at any period of the present century. The recent increase of typhus in the metropolis is accounted for by what are now its recognised causes.

In the following pages it is argued, that we have it in our power, not only to arrest the spread of contagious fevers, but in many cases to prevent their origin. There are still some physicians who refuse to subscribe to the doctrine of spontaneous generation of the poison of any specific disease. It has been maintained with no small ingenuity and learning, that because there is no such thing as spontaneous generation of plants and animals, there can be no such thing as the spontaneous origin of the poison of fever. But, in order to draw a just conclusion from such a comparison, there must be some analogy between the objects compared. Yet a moment's reflection shows, that there exists no analogy between the poison of fever and an organised being. Although we are as yet totally ignorant of the actual nature and composition of fever poisons, and know not whether they be solid, fluid, or gaseous, the possibility of their spontaneous origin is not more surprising than the production of other non-organised products. No mention is

[b] Boudin, 1845, pp. 126-7.

made of specific diseases in the Mosaic account of the Creation, when we are told that every living creature and herb of the field was created, and it would be absurd to imagine that all of them have sprung from Adam. They must, therefore, have been produced at some more advanced stage of the world's history, and, if this be so, their analogy to plants and animals fails, and it is equally reasonable to believe in the possibility of their spontaneous generation at the present day. It is not impossible that, as time rolls on, the mode of origin of other specific poisons, such as those of syphilis, small-pox, and scarlet fever, will be discovered. Lastly, there are certain specific diseases, such as puerperal fever, gonorrhœa, and hydrophobia, which few will deny to be sometimes generated *de novo*, and afterwards propagated by contagion or infection: the spontaneous origin of continued fevers is not more extraordinary.

But discarding altogether the doctrine of spontaneous generation, it must be conceded, that the circumstances which favour the propagation of Continued Fevers vary according to the species of fever, and are equally deserving of study, whether they be regarded as predisposing or primary exciting causes.

There is another subject opened up by a study of the causes of specific fevers, viz., the possibility of two specific poisons producing their effects upon the system at the same time, and thus giving rise to diseases of a more or less hybrid character. It will be shown in this work, that Hunter's doctrine that one specific poison in the system is incompatible with the existence of another is no longer tenable, and that two specific diseases may run an undisturbed course at one time. Here is a new line of investigation, but one that promises to be rich in results.

The term Fever or Pyrexia is employed in two very different senses; first, to express that group of general constitutional symptoms which accompany local inflammations; and secondly, to denote a similar group of symptoms, which, though occasionally complicated with local inflammations, are quite independent of them, and result from the absorption of some poison into the system from without, or from the action on the nervous system of a non-specific cause. In the former case, we say that the fever is *symptomatic*, in the latter, *idiopathic* or *essential*. It is true, that it has been contended that there is no such thing as idiopathic fever, but that fever is always symptomatic of some local lesion. Thus with regard to the Continued Fevers, with which we are more immediately concerned, it was maintained by Broussais, that all Continued Fevers were symptomatic of

inflammation of the gastro-intestinal canal, and by Clutterbuck that they were symptomatic of inflammation of the brain or its membranes. The writings, however, of Graves, Stokes, and Christison, and the labours of modern pathologists have completely demonstrated the fallacy of such views.

It would be more curious than instructive to enter into a consideration of the numerous views, according to which medical writers have endeavoured to explain the phenomena of fever, to show how the humoralists, headed by Hippocrates and Galen, looked upon fever as the result of a contest on the part of nature to expel from the system a superabundance of one or other of the four humours, blood, phlegm, yellow or black bile; how the solidists, represented by Fernelius, Hoffmann, and Cullen, imputed it to changes in the living solids; how, on the one hand, Tweedie insisted that the blood was primarily affected, while, on the other, Christison urged that the first link in the chain of events was derangement of the nervous system; how Brown held that fever was an asthenic state of the system arising from an abstraction of the natural stimuli, or from exhaustion, direct or indirect, of the excitability; how Ploucquet, Beddoes, Clutterbuck, Armstrong, Mills, and Broussais, maintained that fever was always the result of inflammation or congestion.

It is, however, not a little remarkable that modern investigations tend to reproduce, in a more exact form, certain opinions concerning the nature of fever, which were entertained by the earliest writers on medicine. The abstract definition of Fever given by Hippocrates, Galen, and Avicenna was 'Essentia vero 'febrium est præter naturam caliditas,' whilst the definition given by one of the greatest of modern pathologists, Professor Virchow of Berlin, is 'Fever consists essentially in elevation of temperature 'which must arise in an increased tissue-change, and have its 'immediate cause in alterations of the nervous system.'[c] Traube's definition is the same, 'Fever consists essentially in an increased 'temperature of the blood.[d]

It is now universally admitted, that, in all forms of fever, there is an actual increase of the animal heat. De Haen long ago proved by the thermometer, that the temperature is increased even in the cold stage of fever. In one case of pythogenic (typhoid) fever admitted into the London Fever Hospital, the temperature was ascertained to be 108° Fahr., and in all fevers, it exceeds at

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[c] Virchow, 1854; Parkes, 1855; Jenner, 1856.
[d] Traube, 1853.

some period the normal standard. The natural heat of the body arises from the oxidation or combustion of certain components of the food, or of the tissues, and it seems equally certain that the preternatural heat of fever is the result of chemical action exalted above the standard of health. The augmented combustion of the tissues in fever is obvious from the loss of weight which the body sustains—a loss far exceeding what can be accounted for by the mere abstraction of food. But we have additional proof of this exaggerated tissue-metamorphosis in fever, in the increased amount of the products of combustion found in the excretions, and more especially in the quantity of urea contained in the urine.[e] Thus Dr. Alfred Vogel[f] has found the urea increased in one case of pythogenic (typhoid) fever to 890 grains in the day; in another case to 1065 grains, and in a case of pyæmia to 1235 grains, the normal amount being only about 400 grains; and similar results have been obtained by many other pathologists. Moreover, there is found to be, on the whole, a direct relation between the temperature and the amount of urea, although the former is modified according to the amount of fluid evaporated from the surface of the skin. As a rule, the temperature is highest, and the quantity of urea greatest, in the early stages of fever; in the advanced stage, when the heart's action fails, the metamorphosis of tissue is reduced, the temperature falls, and the quantity of urea is usually less than at first.

Not only is the formation of urea increased during fever, but the quantity of uric acid is often doubled, while the chloride of sodium and water of the urine are usually diminished.

It is chiefly the nitrogenous tissues of the body that are consumed during fever. Although there is a decrease in the amount of fat, there is no reason to believe that the rate of its diminution is greater than is accounted for by the state of inanition. The experiments of Malcolm so far as they go, show that, in typhus, the product of the combustion of the fatty elements of the food and tissues—the carbonic acid excreted by the lungs—is diminished rather than increased. Further investigations, however, are still wanting on the changes which the respired air undergoes in fevers.

The large amount of urea developed in continued fevers, may be all eliminated by the kidneys, or a portion may be thrown off by

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[e] Our own countryman, Dr. Prout, long ago pointed out, that the amount of urea formed in the body is always increased during fever, notwithstanding the diminution of food; and many recent observers have added greater precision to this general statement.

[f] *Zeitschrift J. Rat. Med.* Bd. iv., Hft. 3.

other channels, or be retained in the blood, either as urea, or as carbonate of ammonia, the product of its decomposition. Hence it is that occasionally the temperature is elevated without a corresponding augmentation of urea. The urea or carbonate of ammonia, with other products of tissue-metamorphosis, circulating in the blood give rise to symptoms of uræmic poisoning (typhoid symptoms.[g]) Every practitioner must have been struck with the remarkable resemblance between a case of typhus in its advanced stage, and one of uræmia dependent on renal disease; in fact, the two affections are very often mistaken for one another.[h] It is highly probable that the symptoms, in both cases, are due to the

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[g] The exact pathology of uræmia is still a subject of discussion. According to Frerichs, the simple accumulation of urea in the blood will not give rise to the so-called uræmic symptoms, and the real toxic agent is carbonate of ammonia resulting from the decomposition of the retained urea by some ferment in the blood (*Die Brightsche Nierenkrankheit*, 1851). Hammond and Richardson, on the other hand, have more recently supported the old view, according to which the urea itself is capable of exciting uræmic symptoms (HAMMOND, *in Americ. Journ. of Med. Sc.*, January, 1861, and *Edin. Med. Journ.*, Oct. 1861; RICHARDSON'S *Asclepiad*, 1862.) Dr. Oppler, of Berlin, who has lately investigated the subject, opposes the view that uræmic symptoms are due to urea in the blood, because Bright, Christison, and Owen Rees, have shown that urea may exist in large quantities in the blood, without any symptoms of uræmia, and because certain French observers have injected a large amount of urea into the blood without producing any other effect than diuresis. He also objects to Frerichs's theory, because he did not find that the injection of carbonate of ammonia produced the heaviness and drowsiness of uræmia, and because, after extirpating the kidneys and tying the ureters of animals, he found much urea, but no carbonate of ammonia, in the blood. He observed, that when the functions of the kidneys were arrested, products of retrograde metamorphosis (Kreatine and Leucine) were formed and accumulated largely in the muscles, and that the extractive matters of the blood were greatly increased. He concludes that a similar increased metamorphosis occurs in the central organs of the nervous system, and that this chemical change accounts for the symptoms of uræmia. Oppler also adduces experiments to show that the kidneys have the power of transforming kreatine into urea. (VIRCHOW'S *Archiv.* Bd. xxi. Heft. 3.) But, whatever theory be adopted, the clinical fact remains, that the symptoms of uræmia are produced by whatever interferes with the excreting function of the kidneys.

[h] Dr. Richardson has attempted to distinguish between the symptoms produced by urea, and by ammonia in the blood. The typhoid state of continued fevers, he believes to be due to the latter substance, and to differ from true uræmia in the occurrence of jactitations in place of paroxysmal convulsions; in the tendency to the hæmorrhagic diathesis, as evidenced by petechial eruptions and fluxes of blood; and in the absence of prolonged coma, which is the leading symptom of uræmia. I cannot admit the applicability of these distinctive characters in practice. Considerable experience at the London Fever Hospital, where cases of renal disease are constantly being sent in as examples of typhus, leads me to say, that the first and last points of distinction would of themselves afford no aid in diagnosis. The presence of the specific eruption would of course decide in favour of typhus; but, failing the eruption, a certain diagnosis is often impossible. Dr. Richardson admits that the morbid appearances of the blood and internal organs after death are the same in both cases (RICHARDSON'S *Asclepiad*, 1862, p. 191.)

circulation of the same morbid materials in the blood. This is not a mere conjecture. It will be shown, in a subsequent part of this work, that in cases of typhus and relapsing fever, with severe head-symptoms, no lesions are to be found in the brain or its membranes, but that, in several cases, urea has been detected in the blood, while the occurrence of epileptiform convulsions in the former, and of head-symptoms in the latter, is usually preceded by a great diminution in the amount of urine. In pythogenic or typhoid fever also, it will be shown, that the cerebral symptoms have probably a similar origin. It is difficult to say why the urea is excreted in some cases, and retained or decomposed in others; but it is not improbable that its elimination is often prevented by some morbid condition of the secreting tissue of the kidney, either of old standing, or consequent on the febrile attack. Serious disease of the kidneys, indeed, is an almost fatal complication of typhus and of some other fevers. There is also reason to believe, that a sudden check to the excretion of urea and of other products of tissue-metamorphosis, is one cause of the development of local inflammations in the course of fever. Cases of idiopathic fevers have been observed, where a sudden diminution in the amount of excreted urea was followed by an attack of pleurisy or of other local diseases, the quantity of urea again increasing as the local complication receded.[1] It is important to add, that critical deposits are chiefly observed in the urine in cases where there is reason to believe from the symptoms, that the products of tissue metamorphosis have been retained in the system. After convalescence is fairly established, and the patient is regaining weight, the elimination of urea and other products of metamorphosis is found to be diminished below the normal standard.

Although the occurrence of cerebral symptoms, and of the typhoid state in particular, be mainly due to the retention in the blood of the products of tissue-metamorphosis, other circumstances probably contribute to their development, such as the abnormal, or defective nutrition of the central organs of the nervous system, and the non-aëration of the blood consequent on pulmonary complications.

As the metamorphosis of tissue, which occurs during health, is under the influence and control of the nerves, so the augmented metamorphosis which occurs in fever is probably, in a great measure, due to some abnormal condition of the nervous system. According to the beautiful experiment of Claude Bernard, an elevation of temperature to the extent of

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[1] See PARKES, 1855.

from 7° to 11° Fahrenheit, is always produced on one side of the face of an animal, when the trunk uniting the sympathetic ganglia of the neck, on the corresponding side, is divided; the sensibility of the part is greatly excited and the vessels are dilated and hyperæmic, but there is no exudation, œdema, or pathological change of structure. The elevation of temperature in such a case must be referred to the hyperæmia and the increased metamorphosis of tissue in the part, which had before been held in check by the influence of the sympathetic nerve. The converse of this experiment has been performed by Waller, who found that contraction of the dilated vessels, diminution of vascular injection, and reduction of temperature followed the irritation of the divided sympathetic, by the transmission through it of an electric current. Experiments on the vagus nerves have been attended with similar results. Weber ascertained that section of the vagus was followed by increased rapidity of the heart's action, the number of beats being again reduced on passing an electric current through the cut nerve. Volkmann and Fowelin observed that section of the vagus caused an increased lateral pressure of the blood in the arteries, whilst Ludwig and Hoffa found the lateral pressure diminished by irritation of the nerve.[k] These observations make it probable, that the increased metamorphosis of tissue and the elevated temperature in fever are due to a semi-paralysed condition of the sympathetic nerves, although it is possible that they are, to some extent, directly modified or increased, by the altered condition of the blood itself. The impaired nervous force also accounts in part for the liability of the internal organs to become inflamed during fever, while the accelerated action of the heart may be due to paralysis of the vagus.[l]

There are many facts indicating that the nervous system exercises a powerful influence on the early phenomena of fever, such, for example, as the languor and prostration usually complained of from the first, and the occasional occurrence of sudden death at the onset. In Simple Continued Fever, which is independent of a specific poison, the nervous system seems to be affected primarily. The best illustration is to be found in the fever that occasionally results from sheer nervous exhaustion, consequent on mental or bodily fatigue. But, as regards the other continued fevers, which are due to some poison, the poison is probably in the first place absorbed into the blood, and through this medium produces its effect on the nerves. The facts recorded by Sir Henry Marsh and others, to

[k] See *Brit. and For. Med. Chir. Rev.* Ap. 1856, p. 398.

the effect that persons may be seized with symptoms of fever immediately on exposure to the poison, do not prove that the poison acts directly on the nerves, without being absorbed into the blood, for hydrocyanic acid may prove fatal in a few seconds after its application to the tongue, and be afterwards detected in the blood of the heart.[m]

The muscles, being deprived in the manner described of their healthy nervous stimulus, the patient naturally suffers from a feeling of incapacity for exertion or motion; at the same time, the muscular and other tissues begin to waste, the retrograde metamorphosis being favoured by the accelerated circulation of the blood. The blood then becomes contaminated by the *debris* of the disintegrated tissues in addition to the original fever-poison. These morbid materials may be eliminated by the natural channels, and so be productive of no injury; but if there be any impediment to their excretion, they give rise to the consequences already pointed out. When stupor, delirium, and coma present themselves in the course of fever, it is the custom to refer these symptoms to the action of the fever-poison on the brain; but the cerebral functions are more probably deranged, not by the fever-poison, which was the first and necessary link of the pathological chain, but by the accumulation in the blood of the products of tissue-metamorphosis, and by the perverted and defective nutrition of the brain itself. Hence it is, that the symptoms in the advanced stages of many fevers are closely assimilated, although the primary poisons have been perfectly distinct.

According to the present extent of our information, the phenomena of idiopathic fevers, may be summed up, as follows:—

1. The fever-poison enters the blood.
2. The nervous system (and particularly the sympathetic and vagus) is paralysed.
3. The retrograde metamorphosis of the muscles and other tissues is increased, while, at the same time, little or no fresh material is assimilated to compensate for the loss. Increased temperature, great muscular prostration, and loss of weight are the results.
4. This destruction of tissue is increased by the accelerated action of the heart.
5. The non-elimination of the products of tissue-metamorphosis gives rise to cerebral symptoms and local inflammations.

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[m] CHRISTISON, *On Poisons*, 3rd ed. p. 697.

6. On the elimination of the fever-poison and of the products of tissue-metamorphosis, the nerves resume their normal function, the undue consumption of tissue is checked, and the patient regains his strength and weight. It is impossible to say why this termination occurs at a definite time in different fevers.

If this be the correct pathology of fever, our objects in treatment ought to be:—

1. To neutralise the poison and improve the state of the blood.
2. To promote elimination.
3. To reduce the temperature and the action of the heart.
4. To sustain the vital powers by stimulating the paralysed nervous system, and supplying nourishment to compensate in some measure for the increased consumption of tissue.
5. To relieve distressing symptoms.
6. To obviate and counteract local complications.

These remarks afford some idea of the light shed by modern research on the pathology of fever, and show the importance of carefully investigating the effects of disease and of remedial agents on the various excretions. The subject is yet in its infancy; but it is in the direction indicated that we must look for future progress.

CHAPTER II.

Typhus Fever.

Section I.—Definition.

A DISEASE attacking persons of all ages, generated by contagion, or by overcrowding of human beings, with deficient ventilation, and prevailing in an epidemic form, in periods, or under circumstances, of famine and destitution. Its symptoms are: more or less sudden invasion marked by rigors or chilliness; frequent, compressible pulse; tongue furred, and ultimately dry and brown; bowels, in most cases, constipated; skin warm and dry; a rubeoloid rash appearing between the fourth and seventh days, the spots never appearing in successive crops, at first slightly elevated, and disappearing on pressure, but, after the second day, persistent, and often becoming converted into true petechiæ; great and early prostration; heavy flushed countenance; injected conjunctivæ; watchfulness and obtuseness of the mental faculties, followed, at the end of the first week, by delirium, which is sometimes acute and noisy, but oftener low and wandering; tendency to stupor and coma, tremors, subsultus, and involuntary evacuations, with contracted pupils. Duration of the fever from ten to twenty-one days, usually fourteen. In the dead body no specific lesion; but hyperæmia of all the internal organs, softening of the heart, hypostatic congestion of the lungs, atrophy of the brain, and œdema of the pia mater are common.

Section II.—Nomenclature.

Typhus fever has been described under many different appellations. The following are the most important: —

1.—*Typhus.*

Typhus (*Sauvages*, 1759; *Cullen*, 1769); Enecia Typhus (*Mason Good*, 1817); Typhus and True Typhus (*Modern English Writers*).

2.—*Derived from its contagious character.*

Λοιμὸς *pro parte* (Greek writers); Febris pestilens (*Galen? Celsus? Fracastorius*, 1546; *Salius Diversus*, 1584; *Riverius*, 1623; *Willis*, 1659; *Sydenham*, 1668); One of the 'Morbi contagiosi' of Fracastorius (1546); Parish Infection (*English Bills of Mortality*, 1600—1700);

Infectious Fever (*Lind*, 1763); Pestilential Fever (*Grant*, 1775, *Stoker*, 1826); Der ansteckende Typhus (*J. V. Hildenbrand*, 1810); Typhus contagieux (*J. C. Gasc*, 1811); Contagious Fever (*Bateman*, 1818); Tifo contagioso (*Rossi*, 1819); Contagious Typhus (*English Writers.*)

3.—*Derived from its Prevalence in Epidemics.*

Febris Epidemica (*J. Burserius*, 1625); Epidemical Epidemic Fever (*Rogers*, 1734); Febbre Epidemica (*Rasori*, 1813); Epidemic Fever, *pro parte* (*English Writers*).

4.—*Derived from the Cutaneous Eruption.*

Morbus pulicaris (*Cardanus*, 1545); Febris Pestilens quam Cuticulas vel Puncticula vocant (*Fracastor.* 1546; *Forestus*, 1591); Tabardiglio et Puntos (*De Torres*, 1574); Febris purpurea epidemica (*Therœus*, 1578; *Coyttarus*, 1578); La Pourpre (*Early French Writers, P. a Castro*, 1584); Fleckfieber (*Early German Writers, P. a Castro*, 1584); Febris stigmatica (*Early Writers, P. a Castro*, 1584); Febris petechialis (*N. Massa*, 1556; *Sennertus* 1641; *Selle*, 1770; *Burserius*, 1785); Febris maligna pulicaris seu puncticularis (*Pet. a Castro*, 1584); Pipercoorn (*Early Dutch Writers, Forestus*, 1591); Febris peticularis (*Roboretus*, 1592); Morbus puncticularis (*Donkers*, 1686); Febris petechialis vera (*F. Hoffmann*, 1700); Spotted Fever (*Strother*, 1729; *Short*, 1749); Febbre petecchiale (*Rasori*, 1809); Morbo petecchiale (*Acerbi*, 1811; *Palloni*, 1819); Das Fleckenfieber (*Reuss*, 1814); Typhus exanthematicus und Das exanthematische Nervenfieber (*German Writers*); Typhorubeoloid (*Roupell*, 1831); Petechial Fever (*Peebles*, 1835); Petechial Typhus (*auct. var.*)

5.—*Derived from the Presence of Cerebral Symptoms.*

Febris maligna cum sopore (*Riverius*, 1623); Fever of the Spirits (*Quincy*, 1721); Typhus comatosus (*Sauvages*, 1759); Brain Fever (*auct. var.*)

6.—*Derived from Tendency to Prostration.*

Febris asthenica (*var.*); Febris atacta *pro parte* (*Selle*, 1770); Fièvre ataxique, Fièvre adynamique, *pro parte* (*Pinel*, 1798); Adynamic Fever (*Stoker*, 1826; *Burne*, 1828).

7.—*Derived from a supposed Putrid or Malignant Character.*[a]

Febris putrida et maligna, Synochus putris and S. cum putredine (*Old Authors*); Febris maligna pestilens (*Riverius*, 1623; *Sennertus*, 1641; *Willis*, 1659); Febris cacoetes (*Bellini*, 1683); Malignant Fever (*Langrish*, 1735; *Fordyce*, 1791); Febris continua putrida (*Boerhaave*, 1738; *Wintringham*, 1752); Putrid Malignant Fever (*Huxham*, 1739). Febris exanthematica, maligna et venenosa, et perniciosa (*J. F. Bianchini*, 1750); Febris maligna (*Le Roy*, 1771); Putrid Continual Fever (*Macbride*, 1772); Febris continens putrida (*Selle*, 1770); Febris lenta

[a] The terms *putrid* and *malignant* have often been applied to other fevers of a severe or typhoid type.

nervosa maligna (*Burserius*, 1785); Das Faulfieber (*Hecker*, 1809); Febbre putrida (*Ital.*); Fièvres putrides et malignes, *pro parte* (*Fr.*); Typhoid Fever, with putro-adynamic character (*Copland*, 1836).

8.—*Derived from its Prevalence in Camps and Armies.*

Pestis bellica and Typhus bellicus (*var.*); Morbus Castrensis vel Morbus Hungaricus, *pro parte* (*Sennertus and many old authors*); Morbus qui ex castris in Bavariam penetravit (*Rhumelius*, 1625); Febris Castrensis (*Willis*, 1659; *Haller*, 1742); Febris militaris (*Petri*, 1665); Febris Castrensis Petechialis Epidemica (*Brandhorst*, 1746; vide *Haller*, 1758); Typhus Castrensis (*Sauvages*, 1759); Camp Fever (*Grant*, 1775); Die Kriegspest (*Hufeland and Reuss*, 1814) Typhus des Camps et des Armées (*Louis*, 1829).

9.—*Derived from its Prevalence in Prisons.*

Febris contagiosa in carceribus genita (*Huxham*, 1742); Jayl Fever (*Pringle*, 1750; *Heysham*, 1782; *John Howard*, 1784); Typhus Carcerum (*Sauvages*, 1759); Febris carceraria (*Burserius*, 1785); Jail Distemper (*J. C. Smyth*, 1795); Maladie des Prisons (*French writers*).

10.—*Derived from its Prevalence in Hospitals.*

Malignant Fever of the Hospital (*Pringle*, 1752); Febris nosocomialis (*Burserius*, 1785); Fièvre des Hôpitaux (*French*).

11.—*Derived from its Prevalence in Ships.*

Febris pestilentialis nautica (*Huxham*, 1752); Ship Fever (*Lind*, 1763, *Grant*, 1775); Febris nautica (*Burserius*, 1785); Infectious Ship Fever (*Blane*, 1789).

12.—*Derived from its supposed Mode of Origin.*

Ochlotic Fever (ὄχλος, a crowd), (*Laycock*, 1861.)

13.—*Other Synonyms.*

Irish Ague (*Old Irish Designation*); Morbus mucosus (*Roederer and Wagler*, 1762); Catarrhal Typhus (*Irish writers*); Febris inirritativa (*Darwin*, 1800).

The appellation Typhus, originating with Sauvages, adopted by Cullen, and now in general use, is not very appropriate. The word τύφος literally means smoke, but was employed by Hippocrates to define a confused state of the intellect with a tendency to stupor ('stupor attonitus'). In the latter sense, it expresses a prominent symptom in the disease. The expression πυρετὸς τυφώδης, or *Febris typhodes*, as employed by Galen, Prosper, Alpinus (1611), Recalchus (1638), Juncker (1718), &c., did not apply to any specific fever, but had a much more general application. Here is Juncker's definition: '*Typhodes* dicitur, quando inflammatio 'erysipelacea, vel hepatis, vel ventriculi, vel uteri, febrem provocat,

'quæ anxiis, frigidis et inutilibus sudoribus conjuncta est. Deri-
'vatur a τύφος, seu res inanis fumo similis.'[b]

Previous to the time of Sauvages, Typhus was known as Pestilential or Putrid Fever, or by some name derived from the eruption, or expressive of the locality in which it appeared, as Camp, Jail, Hospital, or Ship Fever.

Section III.—Historical Account of Typhus Fever.[c]

TYPHUS Fever is a disease of great antiquity. It was possibly one of the diseases to which frequent allusion is made in the Sacred Writings under the term pestilence, which appeared under the same circumstances—over-crowding and famine—as are now known to give rise to typhus.

Typhus does not correspond with any of the divisions of fever made by Hippocrates, but some of the cases recorded in his book on epidemics closely resemble it.[d]

During the first fifteen centuries of the Christian era, numerous epidemics of contagious fever occurred under circumstances of overcrowding and famine in different parts of Europe, but the descriptions of the Greek, Latin, and Arabian writers, are not sufficiently precise to warrant us in asserting that they consisted of typhus.[e] In many instances, the disease was oriental plague, while in others it was probably typhus. These two affections were long confounded, and the terms *Λοιμὸς*, *Pestis*, and *Febris Pestilens* were applied to both in common. The plague of Athens, which broke out during a siege, when the city was suffering from famine and overcrowding, was possibly typhus. It was contagious, and the attendants upon the sick especially suffered. Dr. Adams, the learned commentator of Hippocrates, believed that the disease

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[b] *Consp. Med. Theor. Prat.* Halæ, 1734, p. 500.

[c] The following history is far from being complete. A complete history of typhus would be the history of Europe for the last three and a half centuries. An imperfect attempt has been made to give some particulars respecting the most famous of the great fever epidemics, to ascertain the actual nature of the fever in each instance, to point out the circumstances under which the epidemic appeared, and to allude to the principles of treatment adopted at different periods. For additional details respecting the history of Typhus, the reader is referred to the works enumerated in the Bibliography, and more particularly to those of Webster, A.D. 1800; Vilalba, 1803; Palloni, 1804 and 1819; Hildenbrand, 1811; Wawruch, 1812; Rasori, 1813; Acerbi, 1822; Schnurrer, 1823; Ochs, 1830; Ozanam, 1835; Gaultier de Claubry, 1838; West, 1840; Hecker, 1844; and Ritchie, 1855.

[d] See, for example, Case XV., in the Third Book of Epidemics. *Syd. Soc. Transl.* Vol. i. p. 419.

[e] For references to the Greek, Latin, and Arabian writers on fever, see Dr. Adams's Translation of Hippocrates (*Syd. Soc. Ed.* Vol. i. p. 339), and of Paulus Ægineta (*Syd. Soc. Ed.* Vol. i. p. 187.)

was bubonic plague,[f] but no mention of buboes is made in the graphic history of Thucydides, which corresponds in most particulars with the typhus that appeared in later times, during the siege of Saragossa. In the works of Livy, Tacitus, and other Roman writers, frequent allusion is made to pestilences which devastated Rome; no account of the symptoms is preserved, but the pestilence usually appeared in seasons of famine, and on one occasion Galen fled from Rome, on account of its contagious character.[g]

In the year 1489, no fewer than 17,000 of the troops of Ferdinand, then besieging Granada, were destroyed by a fever, which the Spaniards, from its spotted character, styled "*El Tabardiglio*," a designation which was afterwards certainly applied to typhus.[h]

The sixteenth century, remarkable for the revival of religion and letters, was likewise noted for the number and severity of its epidemics; and now, for the first time, there is unmistakeable evidence that many of these epidemics were typhus, in the accurate descriptions handed down by two Italian physicians Fracastorius[i] of Verona and Cardanus of Pavia.[k]

Fracastorius (nat. A.D. 1483, ob. 1559), described very minutely the symptoms of an epidemic fever (*Febris pestilens*) that prevailed in Italy in the years 1505 and 1528, its appearance on both occasions being preceded by very inclement seasons, and almost total destruction of the crops. It was contagious and very fatal, and was characterised by an eruption, vulgarly denominated 'Lenticulae' or 'Puncticula.' 'Circa quartum vel septimum diem, in brachiis, 'dorso et pectore, maculæ rubentes, sæpe et puniceæ, erumpebant, 'puncturis pulicum similes, sæpe majores, imitatæ lenticulas, 'unde et nomen inditum est.' The other symptoms were great prostration, feeble pulse, thirst, sordes on the tongue, injected conjunctivæ, blunting of the mental faculties, and, after the fourth or seventh day, mental aberration and low muttering delirium; in some, wakefulness; in others, somnolence; and in others, both of these conditions in succession. The disease lasted from seven to fourteen days, and occasionally longer. Retention of urine, and a deficient or livid eruption were regarded as bad symptoms. A supporting treatment was considered the best, and the majority of those who were bled, perished.[l] The disease was distinguished from the true plague, which was described under the title, '*Febris* '*vere pestilens*.'

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[f] Transl. of HIPPOC. *Syd. Soc. Ed.* vol. i. p. 384.
[g] Transl. of PAULUS ÆGINETA. *Syd. Soc. Ed.* vol. i. p. 281.
[h] VILALBA, 1803, vol. i. p. 69. [i] FRACASTORIUS, 1546. [k] CARDANUS, 1545.
[l] 'Certe res cidit, ut major pars phlebotomorum, perierit.'

That the disease observed by Fracastorius was the typhus of modern times is placed beyond doubt, by the circumstance that the eruption so closely resembled that of measles, that medical men found it necessary to point out the distinctions between the two affections. Cardanus said that one of the greatest errors committed by practitioners of his day was: 'Quod pulicarem morbum mor-'billum credunt.'[l] Nicholas Massa of Venice devoted a chapter to the distinctions between the *petechiæ* of fever, and the eruptions of measles and small pox;[m] and Montuus remarked: 'Sed falso 'morbilli putantur, puncta quædam pulicum morsibus non dissi-'milia, quæ per febres pestilentes in cutis superficie aliquando 'visuntur.'[n]

In the years 1550—54, during a season of great scarcity, and a consequent crowded state of the large towns, a petechial fever prevailed in Tuscany and destroyed upwards of 100,000 persons.[o] About the same time (1552) a similar fever devastated the army of the emperor Charles V., during the siege of Metz, and was described by Andreas Gratioli.[p]

In 1557, typhus was extensively prevalent in France, and formed the subject of an extensive work 'De Febribus purpuratis' by Coyttarus,[q] of Poitiers. Some years later, Ambrose Paré,[r] described a 'pestilential fever' as prevailing in France along with true plague, in which the skin was marked by 'maculæ pulicum 'aut cimicum morsui similes.'

In 1566, the notorious '*Morbus Hungaricus*' appeared in Hungary in the army of Maximilian II, and thence spread over the whole of Europe. It was eminently contagious, and among its symptoms was intense headache, followed by delirium, a dry, black tongue, and occasionally abscesses of the parotid, and gangrene of the extremities. There was likewise an eruption upon the skin in many cases, consisting of spots resembling flea-bites, but differing, as Sennertus pointed out, in the absence of a central punctum. The duration of the disease was from fourteen to twenty-one days.[s]

In 1580, Verona was again the scene of an epidemic of typhus, which was admirably described by Petrus à Castro, under the

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[l] Cardanus, 1545, ed. 1663, tom. vii. sect. 1. cap. 36, p. 216.
[m] Massa, 1556, cap. iv. p. 67—70. [n] Montuus, 1558, lib. vii. cap. 2.
[o] Vide, Palloni, 1804 & 1819; & Peebles, 1835.
[p] Gratioli, 1576; & Ozanam, 1835, vol. iii. p. 127.
[q] Coyttarus, 1578. [r] Paré, 1568.
[s] Sennertus, 1619. Ozanam, 1835, vol iii. Ritchie, 1855. For numerous other references consult Ploucquet's *Repertorium*.

designation, '*Febris maligna puncticularis seu peticularis.*'[t] It was contagious, and prevailed chiefly in the winter months; and, among the causes to which it was referred, was famine. Among the symptoms were frequent, small, weak, pulse; dry, black tongue; vascular injection of the face and eyes; wakefulness and delirium; stupor passing into coma; tremors and subsultus; parotid abscesses, in some cases; and an eruption appearing from the fourth to the seventh day of the disease. This eruption was said to resemble flea-bites, but the points of distinction were noted. The disease, Castro states, was designated '*La Pourpre*' by the French; '*Tabardiglio*' by the Spaniards; '*Petecchie*' by the Italians; and '*Fleckfieber*' by the Germans (p. 45). Bleeding, both general and local, was commended at the beginning of the disease; but, at a later stage, was thought to be dangerous. All the patients exhibited 'ardentissimum vini desiderium, ut continuo 'vinum expostulantes lacessant.' This epidemic appears to have extended over Italy, and formed the subject of another excellent monograph by Salius Diversus of Faenza.[u]

In 1591, Italy was again visited by a severe famine, and an extensive epidemic of contagious fever, which lasted for four years, and was well described by Octavius Roboretus, of Trent, in his work, 'De Peticulari Febre.'[x] The symptoms corresponded precisely with those of the Febris peticularis of Petrus à Castro.

On several occasions, during the sixteenth century, an epidemic of contagious fever prevailed in Spain, which received the name of *Tabardiglio* or *Puntos*, from the spotted character of the skin. Much discussion took place as to whether this fever was identical with true plague or was a distinct malady.[y]

During this century, the first recorded instances occurred in England of the 'black assizes,' to which attention will subsequently be directed.

Petrus Forestus of Alcmaer,[z] in the latter part of the sixteenth century, observed a fever in Holland, then suffering from famine and from the efforts made by the Dutch to throw off the Spanish yoke. This fever was said to agree in every respect with the 'Lenticulæ' of Fracastorius. Speaking of the eruption, Forestus observed, 'Cum vero stigmata latiora essent, et rubedinem habe-'rent, melius evadebant. At nigræ et minutæ, instar piperis nigri, 'lethales erant. Vulgus, a similitudine, appellabant *Pipercoorn*,

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[t] Castro, 1584. [u] Salius Diversus, 1584. [x] Roboretus, 1591.
[y] Vilalba, 1803. [z] Forestus, 1591. ed. 1653, tom I. lib. vi. obs. 35, *et seq.*

'nostro idiomate.' Another symptom of the fever was 'typhomania, 'vel genus delirii cum levi furore mixtum.' In 1635, and again in 1669, the true plague appeared at Leyden and other parts of Holland, and on both occasions was preceded and followed by a contagious 'spotted fever.'[a] Diemerbroeck stated, that in 1635 this petechial fever gradually increased in severity, 'donec tandum in apertissi-'mam pestem transiret.'[b] J. C. Rhumelius,[c] of Munich, published a very curious history of an epidemic of typhus, which originated in 1621, among the confederate troops encamped at Weidhausen, and spread over the whole of Bavaria.

During the thirty years' war (1619—1648), the whole of central Europe was devastated by famine and contagious fever.[d] An excellent description of this fever, as it appeared in the south of France, is given by Lazarus Riverius of Montpelier, under the title of '*Febris maligna pestilens*.'[e] In the city of Montpelier, it broke out during a siege in 1623, and almost one-third of those who were seized, died. The skin was marked by an eruption of red, livid, or black spots, resembling flea-bites, which appeared from the fourth to the ninth day, over all parts of the body, but most frequently on the loins, chest, and neck. As regards treatment, tonics and acids were commended, and wine was often found extremely beneficial: bleeding was never practised, except in very plethoric persons. In 1641, the south of France, and indeed the whole of Europe, were still devastated by typhus, which was celebrated in song by Zylingius.[f]

* * * * 'Per omnes
'Burgundos et quas stagnans Arar irrigat urbes
'Insolita exarsit febris, quæ corpora rubris
'Inficiens maculis (triste et mirabile dictu !)
'Quartâ luce frequens fato pendebat acerbo.'
'Pulsus erat minimus, tremulusque soporque
'Mens vaga, visque labens; lotium crassumque rubensque
'Interdum tenuae instar aquæ.' * * *
* * * *
'Illa eadem Italicæ gentes, miscrumque Sabaudum
'Qui Sequanam, Rhodanumque bibunt, Belgas et Iberum
'Corripuit, necnon Europâ saeviit omni.'
'Accusant alii pluvias, multoque madentem
'Autumnum per flatum austro, qui uligine cœlum
'Corrumpit, fluidæque parit *contagia* pestis

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[a] Webster, 1800. i. 295. [b] Diemerbroeck, 1646. [c] Rhumelius, 1625.
[d] West, 1840, p. 287. [e] Riverius, 1648. [f] Vide Ozanam, 1835, iii. 135.

'Non nulli vitiata putant alimenta malignum
'Suppeditasse homini succum, qui putris adeptâ
'Labe venenatum in venis produxit ichorem.
'Undè venenati morbi, undè et maxima clades
'Obsessos inter cives et agentia castra.
'Sunt qui purpureum hunc morbum *pestemque sequentem*
'Italici sobolem belli regumque duorum
'Gallici et Hispani numerosum militem, et ortas
'In castris febres, * * * censent.'

In the spring of 1643, while the Earl of Essex was besieging the town of Reading, a fever ('*Febris pestilens*,') broke out in the army of the Parliamentary general, and also in the garrison commanded by Charles I; in both armies, the troops were said to have been greatly overcrowded. The fever was accompanied by an eruption of spots, partly red and partly livid. It was contagious, and was communicated to the inhabitants of Oxford and of the surrounding country, and proved very fatal. These particulars are obtained from the account published by Thomas Willis, the celebrated anatomist, then studying medicine at Oxford.[g] Again, in 1658, a fever prevailed over England, which, according to Morton, converted the whole island into one vast hospital. It was contagious, and among its symptoms were a weak pulse, headache, watchfulness or stupor, occasionally subsultus, and an eruption of 'maculæ latæ et rubicundæ morbillis similes in toto corpore.[h]

The great plague of London of 1665, was likewise preceded and followed by an epidemic of malignant Continued Fever (*Febris pestilens*). One of the symptoms of this fever was a red efflorescence on the skin, which in a short time became dark and livid: no buboes were present. Sydenham's description of this fever is mixed up with that of the true plague, and indeed he observed: 'Revera enim cum ipsissima peste specie convenit, nec ab ea nisi 'ob gradum remissiorem discriminatur.' The epidemic appeared at the commencement of 1665, during a season of extreme cold.[i] Sydenham describes another epidemic of Continued Fever ('*Febris nova*'), which commenced in London in the spring of 1685, and extended over the whole of Britain. The two previous winters had been characterised by extreme cold; in that of 1683-4, a fair had been held upon the Thames. This fever presented all the symptoms of typhus: headache and pains in the limbs, dry brown tongue,

[g] Willis, 1659, ed. 1682, p. 113.
[h] Morton. ed. 1696, tom. ii. exercit. 2. Appendix pp. 234-6.
[i] Sydenham, 1685, ed. 1844. p. 95.

delirium and subsultus, and an eruption resembling that of measles, but which was often accompanied by true petechiæ, and was not followed by desquamation.[k]

In 1698, there was a great failure of the crops;[l] and "in October a fatal spotted fever began to prevail all over England."[m]

About the year 1700, F. Hoffmann, professor of medicine at Halle,[n] gave a very accurate description of typhus, under the title of '*Febris Petechialis Vera*,' which he had observed among the German troops in 1683, and which he regarded as very malignant and contagious, and as generated by impure air. Speaking of the eruption, he observed: 'Quarto, quinto, vel etiam septimo die in 'conspectum prodeunt maculæ, in dorso potissimum, et lumbis 'plus minus capiosæ, varii subinde coloris, plerumque tamen sine 'levamine, ideo symptomaticæ magis quam criticæ.' Among the other symptoms were great prostration, severe head-symptoms and delirium, and occasionally gangrene of the extremities. As to treatment, Hoffmann recommended nourishing food, the best wines ('vino nil datur excellentius') and acid medicines. Under the term *Febris Pestilens*, which preceding authors had applied to typhus, Hoffmann described the true glandular plague.

At the commencement of last century, great attention began to be paid in Ireland to epidemic diseases, of which a careful chronological history, extending over a long series of years, is to be found in the writings of Rogers,[o] O'Connell,[p] and Rutty.[q] Typhus, however, was known in Ireland, long before this, under the designation of 'Irish Ague.'[r]

The first epidemic that Rogers observed was at Cork, in 1708. He could not say how long it had existed before, but it appeared to reach its climax in the winter of 1708-9; after that, 'it declined sensibly for a year or two, and then disappeared.'[s] No description is given of this fever, but it is stated that the symptoms were identical with those of the subsequent epidemics of 1718-21 and 1729-31. Short, in his 'History of the Air, Weather and Seasons,' states that the spring and summer of 1707 were the coldest, and the harvest the worst, that had occurred for forty-seven years, (that of 1698 excepted), while the winter of 1708-9 was characterised by 'the greatest frost all over Europe, within 'the memory of man.'[t]

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[k] SYDENHAM, 1685, ed. 1844. p. 488. [l] SHORT, 1749, i. 441.
[m] WEBSTER, 1800, i. 344. [n] HOFFMANN, 1699, ed. 1740, ii. cap. 11, p. 84.
[o] ROGERS, 1734. [p] O'CONNELL, 1746. [q] RUTTY, 1770.
[r] REVIEW, July, 1844, p. 38. [s] ROGERS, 1734, p. 4.
[t] SHORT, 1749, i. 441 and 453.

In 1718, 'a fever, in all respects the same' as that of 1708, became again epidemic in Ireland, and continued until 1721, when 'it abated of its severity, dwindling insensibly away, till at 'length it was rarely to be met with.'[u] It was always most prevalent during the cold months of the year. From O'Connell's description, there can be no doubt that this fever was typhus. The symptoms were headache and anxiety; in some stupor, and in others wakefulness; taciturn, or occasionally vociferous delirium; tremors and subsultus; a dry black tongue, with sordes on the teeth, and an eruption of 'petechiæ rubræ, purpureæ aut lividæ': the duration of the fever was from fourteen to twenty-one days.[x] O'Connell practised venesection under certain conditions; but the counter-indications, respecting which he says, 'a venesectione manum tempero,' were so numerous, as to have precluded the practice from most cases. The rest of his treatment consisted in blisters, salines, and cordials (sal-volatile). A similar fever commenced in York and other parts of England in 1718, reached its acme in July, 1719, and terminated about the close of the latter year.[y] Little is known as to the circumstances under which this epidemic appeared, except that the preceding summer and harvest time of 1717 had been remarkably cold and wet.[z]

After 1721, there was an interval of good health in Ireland, and there was scarcely any fever until 1728, when it returned, after a succession of three bad harvests. Oatmeal, it is stated, rose to an extravagant price, and food of all sorts was so scarce, that riots occurred all over the country, to suppress which, the military were called out. This epidemic lasted four years, and reached its climax in 1731. Rogers attributed the origin of the fever to the same causes as the 'jail fever,' which had appeared at the Oxford and Taunton Assizes. The symptoms, as recorded by Rogers, O'Connell, and Rutty, leave no doubt that the fever was typhus. The tongue became dry and black; the pulse was weak, and there was headache, delirium, and stupor passing into coma. The eruption is well, though quaintly described by Rogers, as follows: 'An universal *Petechial Efflo-* 'rescence, not unlike the measles, paints the whole surface of the 'body, limbs, and sometimes the very face. This appearance is 'very general. In some few, and but few, have appeared *Purple* 'and *Livid Spots*, exactly circular, not unlike those observed

[u] ROGERS, 1734, p. 4. [x] O'CONNELL, 1746, p. 65.

[y] For notices of this epidemic, see ROGERS, 1734; O'CONNELL, 1746; SHORT, 1749; BARKER and CHEYNE, 1821.

[z] SHORT, 1749, ii. 21; ROGERS, 1734, p. 5.

'in the most mortal kind of Small-Pock, some as large as a vetch, 'others not bigger than a middling pin's head' (p. 7, 8.) All the observers mentioned, found that the fever 'did not bear bleeding,' and that a tonic and stimulant treatment was necessary. Rogers recommended sack-whey, wine, salines and blisters. This epidemic was not only general over Ireland, but extended to England. In London, where it was described by Dr. Edward Strother, F.R.C.P., as a 'very remarkable spotted fever,' it proved fatal to many, and in one week raised the bill of mortality to nearly one thousand. The patients had both 'petechiæ and a rash.' In 1728 also, we find from Winteringham, that a fever was prevalent at York characterised by 'red spots, not unlike flea-'bites, on the breast, sometimes interspersed, so that the skin had 'a marbled appearance.' Huxham states that petechial fevers were prevalent everywhere. Although Strother practised bleeding in ordinary fevers, he recommended in this spotted fever a stimulant treatment, consisting of 'warm, moderately strong sack-whey, 'with tea, mutton or chicken broths, water-gruel and wine.'[a]

In 1735, Dr. Browne Langrish, F.R.S., published an excellent account of the fevers prevalent in London in his time. Typhus was described under the term '*Malignant Fever*,' and it was believed to originate from 'the effluvia of human live bodies.' Its principal cause was thought to be overcrowding, with deficient ventilation, as a result of which 'people were made to inhale their 'own steams.' At page 364, the cutaneous eruption is described as follows:—'Petechial spots or red efflorescence in large areas 'sometimes appear upon the skin, and never rise above the surface. 'They seem to be constituted of broken particles of red blood 'oozing from the capillary sanguine arteries, through the lym-'phatic arteries and cutaneous glandules, which, being not minute 'nor subtle enough to perspire through the pores of the epidermis, 'do remain between the epidermis and the cutis in the form of 'flat spots. They do not seem to be critical discharges from the 'blood, because the sick does not grow a whit the better for their 'appearance. The brighter red they are of, so much the better 'sign; but when they appear of a purple brown, or dusky or 'black colour, they manifest a greater degree of putrefaction.' Under the head of treatment, Langrish recommended wine, sulphuric and other acids, and made the following remarks, which

[a] The account of this epidemic has been extracted from SHORT, 1749, ii. 44; ROGERS, 1734, p. 5; O'CONNELL, 1746, p. 268; HUXHAM, 1752; RUTTY, 1770, p. 24; STROTHER, 1729; WINTERINGHAM, quoted by LAYCOCK, 1847, p. 790, BARKER and CHEYNE, 1821, i. p 5.

are well worthy of attention at the present day:—"All medicines 'which strengthen the action of the heart and arteries, and raise the 'pulse, ... without colliquating and dissolving the globules of 'the blood and increasing the alkaline acrimony of the juices, are 'of excellent use.' 'But all the volatile salts and spirits, such as '*Sal. Volat. Succini, Sal. Corn. Cervi, Sp. Sal. Ammon.*, are de-'structive medicines, because they are known to break down and 'colliquate the blood-globules, and to render the animal juices 'more acrid and alkaline.'"[b]

The first edition of Huxham's celebrated 'Essay on Fevers,'[c] appeared in 1739. Chapter viii. is entitled:—'Of Putrid, 'Malignant, Petechial Fevers,' and contains one of the best descriptions of Typhus that had yet appeared. He regarded the disease as contagious, and described both petechial spots, and a measly efflorescence. 'The eruption of the *petechiæ* is uncertain; 'sometimes they appear the fourth or fifth day, sometimes not 'till the eleventh, or even later.' 'The more florid the spots are, 'the less is to be feared.' 'We frequently meet with an efflo-'rescence also, like the Measles, in malignant Fevers, but of a 'more dull and lurid hue, in which the skin, especially in the 'breast, appears as it were marbled or variegated.' Huxham recommended bleeding, provided the patient was very plethoric, and seen at the commencement of the attack; but in most cases, he placed the greatest reliance on bark, mineral and vegetable acids, and generous red wine.

'Petechial Fever' was unusually prevalent in Ireland in the spring of 1735, and in 1736; in connection with this, it may be observed that the years 1734 and 1735 were very rainy, and the 'summers were like winters."[d] After 1731, however, there was no great epidemic of fever until 1740. The winter of 1739-40 was one of intense severity, both in Great Britain and in Ireland. Numbers of cattle and poultry perished of the cold, which also destroyed all vegetable products, and especially the potatoes. The surplus produce of the preceding season having been all exported, a great scarcity followed, so that wheat was sold for 44*s.* the kilderkin, although the same quantity, two years later, fetched only 6*s.* 6*d.* There was great distress among the poor, and many died of starvation. O'Connell's words were:—'Et, quod adhuc funes-'torum malorum cumulum multo gravius adauxit, radices istæ 'tuberosæ (battata vulgo dictæ), nutrimentum fere constans et

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[b] Langrish, 1735, pp. 364 and 369. [c] Huxham, 1739; see also Huxham, 1752.
[d] Rutty, 1770. Pref. p. 33.

'integrum plebeculae et inferiorum hujus regni incolarum, a 'dirissimo hoc et diuturno gelu penitus putrescebant. Hinc 'funesta anonæ charitas, et inter pauperes populumque inferi- 'riorem immaniter sæviens dira fames; hinc putrida plebeculae 'alimenta, ex pravis et corruptis istis radicibus, aliis pravi succi 'vegetabilibus, et morbidorum animalium cadaveribus conflata' (page 325). In August, 1740, an epidemic of fever arose and raged over the whole of Ireland, but particularly in the province of Munster. The epidemic continued throughout the summer of 1741, but towards the close of the year began to abate; in the winter of 1742, after an abundant harvest, it almost completely disappeared. The fever attacked the poor first, but from them it spread to the rich. O'Connell computed that in 1740-41, Ireland lost at least 80,000 inhabitants by famine and spotted fever, and that one-fifth of the population of Munster, where the poor were worse provided for, perished. The fever was characterised by a 'measly rash,' and by the ordinary symptoms of typhus. It is important to notice, however, that there is evidence in Rutty's description of the co-existence of Relapsing Fever with Typhus. This circumstance must be borne in mind, when we read that in some of the cases the pulse was full and hard, and that bleeding was of service,—a statement which must be viewed in connection with the fact, 'that many of the poor, abandoned through neces- 'sity to a low acescent diet, and some of them drinking nothing 'but water, recovered.' In the worst (Typhus) cases, it is stated that bleeding was of no service, and that the pulse was so depressed, as not even to be raised by 'generous cordials and great 'plenty of sack.' Short says that in Galway 'blisters and bleed- 'ing had made doubly fine work of it.' O'Connell strongly condemned much bleeding; and although he bled to ten ounces at the commencement of the complaint, he honestly acknowledged that the treatment was of no use. About the same period, although a little later, a very fatal epidemic fever made its appearance in England, and there are records of its prevalence in London, Bristol, Worcester, Plymouth, etc. In Bristol and Worcester it was observed in 1740, but in London, not until July, 1741. In London it is said to have broken out among the poor who had been half starved for two years, and obliged to eat uncommon and unwholesome things. In all the accounts, mention is made of the eruption; in some cases, it is described as like that of measles, in others as like so many small fleabites, while in a few instances it is said to have been mixed up with petechiae and vibices. Parotid

abscesses and buboes are mentioned by Huxham as frequent complications. In an anonymous pamphlet, published at the time, the treatment recommended consisted in bleeding and purging; but the experience of most observers was opposed to bleeding. Dr. Wall treated his cases with bark and acids; and, in reference to bleeding, he wrote, 'As to myself, I lay so little stress upon 'bleeding, that I have always omitted it, unless some very urgent 'symptom seemed to require it.' Short tells us that the cases in London 'could not bear bleeding.'[e]

In 1750, and again in 1752, Sir John Pringle, Physician-General to His Majesty's Forces, and afterwards President of the Royal Society, described Typhus as 'the Hospital or Jayl Fever.' As to the eruption he wrote as follows:—'There are certain spots, 'which are the frequent, but not inseparable attendants upon fever.' 'They are the true *petechiæ*, being sometimes of a brighter or paler 'red; at other times of a lurid colour; and are never raised above 'the skin. They are small, and commonly distinct; but sometimes 'so confluent, that at a little distance the skin looks only somewhat 'redder than ordinary; but upon a nearer inspection the interstices 'are seen.' 'They sometimes appear as early as the fourth or fifth 'day.' 'The nearer they approach to a purple, the more ominous 'they are.' From the account of the *post-mortem* appearances, however, it is obvious that Pringle included under Hospital Fever, cases which were not Typhus, and which, in fact, were probably not fever at all. Under treatment, he ordered that the patient should first be removed out of the foul air. Speaking of depletion, he observed—'Large bleedings have generally proved fatal, by 'sinking the pulse and bringing on a delirium;' and again: 'Many 'have recovered without bleeding, but few who have lost much 'blood.' He commended bark and serpentaria, and thought 'there was nothing comparable to wine, whereof the common men 'had an allowance to half a pint a day.' Concerning the cause of the fever, Pringle wrote as follows: 'The hospitals of an army 'when crowded with sick . . . . or at any time when the air is 'confined, produce a fever of a malignant kind and very mortal. 'I have observed the same sort arise in foul and crowded barracks; 'and in transport ships, when filled beyond a due number and 'detained long by contrary winds, or when the men were kept at 'sea under close hatches in stormy weather.'[f]

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[e] For an account of this Epidemic, see O'Connell, 1746; Short, 1749; Anonym. 1741; Rutty, 1770; Huxham, 1752; Barker and Cheyne, 1821, i.

[f] Pringle, 1750 and 1752, pp. 291, 301, 317, 326.

Towards the end of 1757, Typhus appeared at Vienna, and lasted till 1759. An account of this epidemic was written by Storck[g] and Hasenöhrl.[h] The disease principally prevailed in overcrowded localities. The pulse was always soft, and the blood drawn in many cases, even at the commencement of the illness, did not coagulate. Although Hasenöhrl recommended venesection in certain cases, he allowed that it was but an 'anceps auxilium.' He spoke, however, in the highest praise of nitric and sulphuric acid, and of the stupendous virtues of Peruvian bark. Storck noted a fatal case, complicated with gangrene of the nose and abscesses of both parotids.

In 1763, Dr. James Lind, physician to Haslar Hospital, published 'Two Papers on Fevers and Infections,'[i] in which he showed that Typhus fever was then a very common disease on board ship, especially during the long voyages from North America. He considered bleeding injudicious, and very often dangerous, treatment in Typhus.

In 1764, a dreadful epidemic of Typhus and dysentery raged at Naples, which was attributed to a great scarcity of provisions, and the consequent starvation and misery of the poorer classes, to whom the disease was for the most part confined. The people from the surrounding country flocked into the city, where they had so few opportunities for attending to the cleanliness of their persons, and were so overcrowded, that their garments are described as saturated with a most offensive effluvium.[k]

After the epidemic of 1740-41, there was but little Typhus in Ireland until 1771. Early in the summer of that year, we learn from Dr. James Sims, of Tyrone, that a fever appeared, which, as autumn advanced, raged with great violence, and lasted for about a year. It was contagious, and was characterised by constipation, soreness of the eyeballs, headache and oppression; about the fourth day, delirium and watchfulness; and in the latter stages, picking of the bed-clothes, pupils insensible to light, black tongue, sordes on the teeth, and involuntary stools. There were also petechiæ of a yellowish colour, with a black speck in the centre. The disease lasted about a fortnight. Bleeding was injurious, and the author recommended acids, free exposure to cold air, bark in large doses, small beer, and claret. The fever prevailed principally among the poor; and its chief cause was considered to be 'a number of persons being crowded together in 'foul confined places.' Dr. Sims considered this fever as 'entirely

[g] Storck, 1761.
[h] Hasenöhrl, 1760.
[i] Lind, 1763.
[k] Sarcone, 1765, pp. 256, 314, 344.

'different' from the low nervous fever of Huxham, which had been prevalent for some years before.[l] Webster tells us, that in the year preceding this epidemic, there was a failure of the potato crop in Scotland, great inundations, and extensive mortality among the cattle in England, but he does not refer to Ireland.[m]

In 1775, Dr. William Grant published 'An Essay on the Gaol, Hospital, Ship, and Camp Fever,'[n] to which I shall subsequently have occasion to allude. From the description, it is obvious that typhus is referred to; the origin of the disease was attributed either to the concentrated emanations from living bodies or to contagion; and as to treatment, it is stated that the antiphlogistic method did not succeed.

In 1780, an outbreak of typhus occurred among the Spanish prisoners confined at Winchester, of whom 268 died in $3\frac{1}{2}$ months. Dr. J. Carmichael Smyth, Physician to H. M. George III, wrote an account of this outbreak, and observed: 'That it arises from 'the putrefaction of the perspirable matter, admits of every species 'of evidence applicable to a matter of fact and observation.' He condemned the use of bleeding as 'highly injudicious, hazardous and often fatal;' and he recommended wine and bark in every stage of the disease. On one occasion, he gave two bottles of port in twelve hours to a patient who recovered; and in other cases, he ordered two bottles of Madeira daily for several days. 'Nothing surely,' he adds, 'can be more absurd, than to use any 'means to diminish the strength of the body, when we are certain 'that sooner or later, the strength will fail and require being 'supported, and when, though the pulse may not be very sensibly 'sunk, there are the most evident signs of debility and 'dejection.'[o]

In 1781, an epidemic of typhus occurred at Carlisle, which will be referred to hereafter. Dr. Heysham,[p] who described the disease, considered it to be one of great debility, and treated all his patients with bark and plenty of port wine.

Rasori[q] has recorded an epidemic of typhus which occurred at Genoa in 1799—1800, when the garrison was besieged by the French, and half famished. The fever was eminently contagious, and was characterised by great prostration, weak pulse, watchfulness and restlessness passing into drowsiness, dry tongue, sordes, very confined bowels, and an eruption 'not very unlike petechiæ,'

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[l] Sims, 1773. [m] Webster, 1800. i. 422. [n] Grant, 1771 and 1775.
[o] Smyth, 1795, p. 81. [p] Heysham, 1782. [q] Rasori, 1813.

which indicated danger according to its abundance. Rasori followed his favourite practice of giving tartar emetic.

At the end of the last, and the beginning of the present, century, another epidemic of typhus made its appearance in Ireland. It commenced towards the close of 1797, reached its acme in 1800 and 1801, and did not terminate until 1803. The period in question was, in Ireland, one of great calamity. The country, for some time before, had not only been threatened with foreign invasion, but had been convulsed by internal rebellion. The upper and the lower classes espoused opposite political opinions, and were arrayed against each other. The consequence was, that the management of the large estates fell into the hands of agents who knew little about the tenantry, many of whom were deprived of employment. To complete the distressing history, there was a succession of bad harvests. An uncommon quantity of rain fell during the summer and autumn of 1797, which injured the crops. The three following years were equally unfavourable, and a great deficiency of the usual supply of nourishment to the poor ensued. The price of bread, potatoes, and indeed of every necessary of life rose enormously. In Dublin, the servants of the upper classes were not allowed potatoes, and bread was portioned out to them sparingly; few persons had more than a quartern loaf in the week. The poor pawned their clothes, and even their bedding for money to purchase food, and, as a natural consequence it was common for several members of one family to sleep in the same bed. As a proof of the great prevalence of the epidemic, it may be stated, that during the two years 1800 and 1801, there were as many deaths from fever in the Dublin House of Industry, as during the next great epidemic of 1817—19. Throughout the epidemic, it was chiefly the poor who suffered; but in proportion to the number attacked, the fever was most fatal among the middle and upper classes. In 1801, there was an unusually abundant harvest, and the poor were again furnished with provisions of all kinds at a moderate price: the epidemic immediately began to decline, and by the end of the following year it had well nigh disappeared. The epidemic spread to England but was less prevalent there than in Ireland. The fever was mainly typhus, although in Ireland relapsing fever was also observed. It was described as highly contagious, and as characterised by the presence of petechiæ and by great debility. Dr. Willan bemoaned the tendency of some physicians in London, to regard the fever as resulting from inflammation of the brain;

and added, 'whoever is bled largely from the arm is precipitated 'to certain death."[r]

It was mainly in consequence of the fever prevalent at this time, that numerous hospitals for the separate treatment of Fever cases, were first established throughout the country. The first was opened at Chester, and its origin was due to the able advocacy of Dr. Haygarth. Liverpool, Manchester, Norwich, Hull, Dublin, Cork, Waterford, and London soon followed this example, the London Fever Hospital being established in 1802.[s]

During the first fifteen years of this century, typhus committed great ravages in the armies of Napoleon, and among the populations of the countries which were the seat of war. It always arose under circumstances of misery and privation, and was particularly prevalent and fatal among the inhabitants of besieged cities. Witness, for example, the melancholy histories of the sieges of Saragossa[t] and Torgau,[u] and of the typhus which ravaged the overcrowded garrisons of Dantzic[x] and Wilna[y] in 1813, and which told with such awful severity upon the famished French troops during the retreat from Moscow, in 1812-13. Numerous accounts of this fever were published at the time, a notice of which will be found in the 13th volume of the Edinburgh Medical and Surgical Journal.[z] Among them, was a most able memoir, by Hildenbrand,[a] on an epidemic of typhus, which prevailed at Vienna, during the winter that followed the campaign of 1806. Hildenbrand maintained that true contagious typhus could be generated by air highly charged with human exhalations. He described the eruption as an *exanthem*, due to dilatation, and rupture of the cutaneous capillaries, and presenting a marbled appearance. He regarded the disease as essentially asthenic; and, although in a few cases he practised bleeding at the commencement, he thought bleeding in most instances useless, or positively injurious. After the first week he had recourse to wine, camphor, and diffusible stimulants; and he adds, that some practitioners were in the habit of prescribing tonics and stimulants from the commencement of the disease. Hufeland[b] also states, that the typhus which appeared in Russia and Poland, during the campaign of 1806-7,

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[r] Consult BARKER and CHEYNE, 1821, vol. i. p. 9 to 20; and WILLAN, 1801, p. 284, for an account of this epidemic.

[s] Consult HAYGARTH, 1801; STANGER, 1802; CLARK, 1802; and Reports of London Fever Hospital.

[t] GAULTIER DE CLAUBRY, 1838, ed. 1844, p. 33. [u] Ib. p. 43. [x] Ib. p. 41.

[y] OZANAM, 1835, iii. 201. [z] *Review*, 1817. [a] HILDENBRAND, 1811.

[b] HUFELAND, 1814. See also HECKER, 1809; REUSS, 1814; ACKERMANN, 1814; and RICHTER, 1814.

was the sequel of hunger, want, and misery; and that in its treatment, antiphlogistics, which were first employed, were found to be unsuitable, and often obviously hurtful. Similar testimony is borne by Baron Larrey: 'La saignée préconisé et mise en pra-'tique par quelques médecins dans cette épidémie, a été constam-'ment funeste.'[c]

In the spring of 1809, typhus made its appearance among the troops landed in England after the retreat from Corunna, and was described by Sir James M'Gregor[d] and Mr. Hooper.[e] Its origin was attributed to the mental depression of the men, and to their being overcrowded on board ship. Wine, brandy, and bark, constituted the treatment recommended by Hooper.

True typhus was epidemic in Italy, in 1816-17, and was well described by Palloni and Rossi.[f] Palloni, like Hildenbrand, insisted on the propriety of classifying it with the exanthemata.

Since the peace of 1815, typhus has often been observed in different parts of Europe.[g] The following remarks, however, will be mainly confined to those epidemics which have occurred in Great Britain and Ireland. Some idea of the varying prevalence of Continued Fevers in these islands may be found from the returns of different hospitals, collected in Table I.

The first great outbreak of fever after 1803, was the epidemic of 1817-19 (see Table I). Of this epidemic, we possess the most ample records for Ireland, in the Reports of Barker and Cheyne, and of Harty; and for Britain, in the works of Bateman, Welsh, and many others.[h] The circumstances under which this epidemic made its appearance, were the following:

1. The winters of 1813-14, and of 1815-16, were of intense severity. In February, 1816, the thermometer, in London, fell in one day to five degrees below zero of Fahrenheit, and during four days it never rose to freezing point. In Ireland, the temperature was not so low; but even there, the cold during the winter and spring of 1815-16 was unusually severe.

2. The winter of 1815-16, was followed by a cold and wet summer and autumn, and in Ireland there was a complete failure of the harvest and of the potato crop. In the neighbourhood of Edinburgh, the crops were still quite green at the beginning of

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[c] LARREY, 1812, ii. 341. [d] M'GREGOR, 1809. [e] HOOPER, 1809.

[f] PALLONI, 1819; and ROSSI, 1819.

[g] See GAULTIER DE CLAUBRY, 1838; VIRCHOW, DÜMMLER, etc., 1849; FORGET, 1854; SCRIVE, 1857; BAUDENS and JACQUOT, 1858; BARRALLIER, 1861.

[h] See Bibliography, 1818—1821.

Table I, showing the Prevalence of Continued Fevers, during the last Forty-five Years.

Years.	DEATHS.		ADMISSIONS.						
	England and Wales.[a]	London.[a]	London Fever Hospital.	Edinburgh Royal Infirmary.[b]	Glasgow Royal Infirmary.[c]	Aberdeen Royal Infirmary.[d]	Dublin. Fever Hospital, Cork Street.[e]	Cork Fever Hospital.[f]	Stockholm Seraphim Hospital.[g]
1817	..	..	760	485	714	..	6,542	3,440	..
1818	..	..	599	1,546	2,336	..	25,502	6,054	.
1819	..	..	524	1,088	1,594	..	3,873	2,398	..
1820	..	..	437	638	289	..	2,994	1,117	..
1821	..	..	232	327	234	..	2,976	1,047	..
1822	..	..	246	355	229	..	2,300	1,159	..
1823	..	..	283	102	269	..	2,668	1,596	..
1824	..	..	444	177	523	..	4,679	1,405	..
1825	..	..	503	341	897	..	4.353	2,809	..
1826	..	..	582	459	926	..	10,612	4,341	..
1827	..	..	611	1,875	1,084	..	6,500	4,312	..
1828	..	..	534	2,013	1,511	..	2.964	1,381	..
1829	..	..	472	771	865	..	3,170	838	..
1830	..	..	475	346	729	..	3,170	1,024	..
1831	..	..	619	758	1,657	..	3,602	1,278	..
1832	..	..	444	1,394	2,733	..	3.991	2.183	..
1833	..	..	249	878	1,589	..	3.332	840	..
1834	..	..	360	690	2,003	..	4.524	1,196	..
1835	..	..	250	826	1,359	..	4.672	1,924	..
1836	..	..	264	652	3,125	..	5.585	4.076	.
1837	..	..	896	1,224	5,387	..	6,595	3,163	..
1838	18,775	4,078	976	2,244	2,228	515	4.042	1.585	..
1839	15,666	1,819	530	1,235	1,529	1,200	5.358	1,970	..
1840	17,177	1,262	234	782	3.385	575	4,329	2.441	211
1841	14,846	1,151	250	1.372	2.578	437	2,872	1.467	326
1842	16,201	1,174	252	842	1,194	282	2,375	1,225	443
1843	..	2,083	1,385	2,080	3,467	1,280	2,529	1,162	238
1844	..	1,696	578	3,339	1,468	780	2,863	1.340	222
1845	..	1,301	477	683	535	378	2.954	2,799	199
1846	..	1,796	506	693	1,565	377	4.555	3,262	518
1847	30,320	3,184	1,259	3,688	5.244	683	5.875	5,693	132
1848	21,406	3.584	967	4,693	1,515	1.648	2,472	1,249	104
1849	17.902	2,482	401	726	570	584	2,977	2,565	66
1850	14,296	1,929	361	520	597	255	2.096	1.756	284
1851	17,121	2,140	614	959	1,385	218	2,133	2,307	443
1852	17,845	2,020	561	691	1,721	148	2.354	1,731	..
1853	18,013	2,483	787	574	1,938	121	1,383	1,643	..
1854	18,332	2,694	714	168	1,058	304	2.069	1,096	..
1855	16,032	2,342	622	201	656	345	2,204	907	..
1856	15,398	2,621	1,300	180	591	225	1,606	1,067	..
1857	18,249	2,096	561	126	546	145	1,466	827	..
1858	17,883	1,865	239	114	370	158	1.310	595	..
1859	15,877	1,789	258	174	311	81	1,616	587	..
1860	13,012	1,392	151	153	357	77	1,478	460	..
1861	..	..	296	141	599	96	..	608	..

[a] Registrar-General's Reports. The term 'typhus' in these reports includes all continued fevers, and probably many cases of other acute diseases.

[b] From a paper by Dr. Christison, 1858, and from private sources. Previous to 1826, the annual report was made up to Dec. 31st, but afterwards, to the end of September, so that the number for 1826 represents only nine months.

[c] Compiled from Cowan, 1838; Orr, 1846 and 1848; M'Ghie, 1855; and private communications from Dr. M'Ghie.

[d] From the Annual Reports of the Infirmary.

[e] Communicated by the Registrar of the Hospital. Up to the end of 1831, the Hospital year terminated on January 4th of the following year; afterwards on March 31st of the following year, so that the entry for 1832 includes five quarters. The numbers for the first two years (1817-18) include the admissions into the 'House of Industry.'

[f] Communicated by Dr. M'Evers, of Cork. In this and the Dublin Hospital, the numbers include all cases of acute disease.

[g] Huss, 1855, p. 29.

September. The harvest of the following year was no better. In September, 1817, the thermometer, in Ireland, fell suddenly from 75° to 30°, and the cold completely destroyed the potato crop, and the late oats; in the month of December, sheaves of corn might be seen rotting upon the ground. Owing to the wet seasons also, the turf or peat, the chief fuel of the poor in Ireland, could not be cut or dried for use.

3. As always happens under such circumstances, many of the working classes were thrown out of employment.

4. Extreme distress ensued. The four-pound loaf was sold in Dublin, in 1817, for 1*s.* 9*d.*; and the poor throughout Ireland are described as wandering about the country gathering nettles, wild mustard, and other weeds, to satisfy the cravings of hunger. This scarcity commenced in the autumn of 1816, and continued until after the harvest of 1818, which was plentiful.

5. During this period commenced, on a great scale, the migration of the poorest classes of Irish into the great towns of England and Scotland, condensing their population, and introducing habits of uncleanliness and improvidence with the seeds of disease.

The epidemic commenced in Ireland, and from thence spread to Britain. Fever first became very prevalent in Cork towards the end of 1816, among a number of operatives who had been thrown out of employment after the conclusion of peace in the preceding year; but the epidemic did not reach its height there until the summer of 1818. In the spring of 1817, the fever began to spread very extensively in Ulster, Munster, and Connaught; but in Leinster, not until the autumn of that year. In Dublin, it commenced in September, 1817. In the autumn of 1819, the epidemic began rapidly to decline, first in Ulster, and afterwards in other parts of the country; and by the end of 1819, the prevalence of fever had almost been reduced to its normal standard. In London, the epidemic commenced in March, 1817; while in Edinburgh, it first appeared in the neighbourhood of the Stockbridge during the following autumn, and rapidly spread.

The probable population of Ireland at this time was, in round numbers, 6,000,000, and the number of sick was estimated at 737,000, or at about one-eighth. In Dublin alone there were 70,000 cases, making about one-third of the inhabitants. According to Donovan, the total number of deaths in Ireland amounted to 44,000. In London, the fever does not appear to have been so prevalent, the total number of cases treated at all the hospitals and dispensaries amounting to only 3,000. In Glasgow, the number of fever cases in the infirmary was 2,715, although for twenty

years before it had never exceeded 130 in the year. In Aberdeen, the total number of fever cases was 2,400.

But, although many cases of Typhus were observed during this epidemic, the fever which mainly characterised it, in Ireland and Scotland at all events, was Relapsing Fever. The reports of Welsh, Harty, Barker and Cheyne, fully bear out the truth of this statement. Welsh remarks, that it was rare to see a fever patient during this epidemic with a measly eruption, that petechiæ were present in only one of fifteen cases, and that relapses were extremely frequent. Dr. Christison, who also observed this epidemic, tells us: 'A true unmistakeable typical typhus, as all 'physicians have understood it in this country since the days of 'Cullen, could scarcely be said to form part of that epidemic.'[i] Relapsing Fever, it must be observed, presents a marked contrast to typhus, not only in its symptoms, but also in its small rate of mortality. In Ireland, out of the 100,337 cases (treated in hospital, and probably the most severe) collected by Barker and Cheyne, only 4,349, or 1 in 23 died; and of 7,608 cases treated in the Dublin Fever Hospital, the deaths were only 258, or 1 in 30½; whereas the mortality in true typhus is about 1 in 5. Of 743 cases observed by Welsh at Edinburgh, only 34, or 1 in 22 died. Throughout Ireland, however, it is everywhere stated, that although the fever was mainly confined to the poor, the rate of mortality was much greater among the rich, being as high as 1 in 5, or 1 in 3. It is doubtful if the proportion of relapsing cases was as great in London as in Ireland; but it is clear from the writings of Bateman, that it was considerable.[j]

The circumstance that the fever in this epidemic was, for the most part, not true typhus, and that it was far from being mortal, must be borne in mind, when we consider the treatment for Continued Fever, which about this time began to be so greatly vaunted, and which for many years continued to exercise much influence over the minds of physicians, if not over the bills of mortality.

At the beginning of this century appeared the works of Ploucquet[k] and Clutterbuck,[l] who endeavoured to show that Continued Fever was a pyrexia, symptomatic of local inflammation of the brain; while about the same time Broussais attempted to localise fever in the bowels, and Beddoes maintained that Continued Fever was

[i] CHRISTISON, 1858, p. 584.

[j] The works from which the account of this epidemic has been derived will be found in the Bibliography, for the years 1818-19-20 and 21. See also STOKER, 1826 and 1835, and DONOVAN, 1848.

[k] PLOUCQUET, 1801.

[l] CLUTTERBUCK, 1807.

always an inflammation, though of variable seat. A fatal blow was struck at the practice of stimulation in typhus, which had been followed from time immemorial, and which had latterly increased in favour, and been carried to a greater extent from the promulgation of the doctrines of Bruno. Although the morbid appearances on which the opinions of Clutterbuck and other writers were founded, are now known to have been fallacious, it was not long before these opinions and the practice which flowed from them were widely adopted. Copious depletion in all forms of fever became the order of the day.

One of the first physicians who carried the views of Clutterbuck into practice on a large scale, was Dr. Mills, of Dublin.[m] In 1812 he treated by venesection (though seldom to more than 6 ounces) 504 cases of 'fever,' of whom only 18 died, or 1 in 28. But an examination of the records of these cases renders it doubtful if many of them were real typhus. Many of them were cases of Relapsing Fever, or a fever of only a few days' duration, and followed by a relapse, while others were examples of enteric fever, ague, pericarditis, or other local inflammations. Although many of the cases are said to have presented 'petechiæ,' yet when 2 only of 73 such cases died, it may be doubted if the 'petechiæ,' which are not described, were those of typhus. We are informed by contemporaneous writers, that fever with an eruption was then far from common.[n] But Dr. Mills's treatment was not so successful as he represented. A complete refutation of his statements appeared in the 'Edinburgh Medical and Surgical Journal,' for July, 1814, in the form of a letter, addressed to the committee of management of the Cork-street Fever Hospital, and signed by the four physicians. Dr. Mills had only been appointed temporary physician for four months, from June 21st, 1810, and for eight months, from April, 1812. He made out that the mortality of his cases during these periods, was much smaller than the average mortality during the previous eight years, of the cases under the care of the other physicians, who had not bled, and that convalescence was likewise more rapid. But it was shown that the mortality had varied very greatly *from year to year*, and that Dr. Mills's results were actually less favourable than those of the other physicians, *at the same time*. Of 709 cases under Dr. Mills, 55, or 1 in $11\frac{2}{13}$ died; whereas of 1,531 cases under the other physicians, 110 died, or about 1 in 13.[o]

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[m] Mills, 1813. [n] *Edin. Med. and Surg. Jour.* vol. vii. p. 435.
[o] See also Stoker, 1835, p. 16.

Four years later appeared the work of Armstrong,[p] who maintained that typhus was, in most cases, accompanied by inflammation, or congestion of the internal organs, and who advocated depletion with much greater energy than Clutterbuck, and practised it in much larger quantities than Mills. Armstrong's practice was widely adopted in the epidemic that immediately followed the publication of his work.

In 1819, Welsh published the results of his observations on the epidemic at Edinburgh,[q] and strenuously advocated the propriety of blood-letting in fever. The average quantity of blood taken from the arm in all Welsh's cases was 24 ounces, but in many the quantity far exceeded this. One patient, a man aged 25, lost 136 ounces at seven bleedings, besides having ten leeches applied. Welsh's cases were almost exclusively Relapsing Fever. With regard to Ireland, Dr. Stokes observes: 'I remember when I 'was a student of the old Meath Hospital, there was hardly a morn-'ing that some twenty or thirty unfortunate creatures were not 'phlebotomised largely. The floor was running with blood; it 'was difficult to cross the prescribing hall for fear of slipping. 'Patients were seen wallowing in their own blood, like leeches 'after a salt emetic.'[r] 'Bleeding,' wrote Dr. Sandwith, of Bridlington, 'was by far the most efficacious agent in the 'treatment; in all cases in which recovery took place without 'bleeding, it was to be regarded as an escape rather than a cure.'[s] The words in which Dr. Bateman, of London, recorded the change in his practice, are so remarkable, as to deserve repetition. In his work, published in 1818, giving an account of the prevailing epidemic, the following passage occurs: 'The other active 'remedy which I have mentioned as capable of abridging the 'course of fever, if employed early, is blood-letting. I believe 'there are few physicians, who, like myself, commenced their pro-'fessional career, impressed with the doctrines that prevailed in 'the schools at the close of the past century, in which the terror 'of debility was certainly predominant, who will not acknowledge 'that their subsequent practice has been a continued struggle 'between the prejudices of education, and the staring conviction of 'opposing facts, which were continually forcing themselves upon 'their observation, and that they have more especially been com-'pelled to a gradual, but material change in their views respecting 'the use of the lancet, not only in fever, but in other diseases. I

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[p] Armstrong, 1816. [q] Welsh, 1819. [r] Stokes, 1854.
[s] Sandwith, 1821.

'am fully convinced of the extent to which my own practice has 'been cramped by this prejudice, and of the reluctance with which 'I have admitted the evidence of my senses, till frequent repeti- 'tions, and the sanctions of other authorities had rendered it irre- 'sistible. My testimony on this point, therefore, cannot be deemed 'the result of haste or temerity.'[t] The change in Bateman's opinion as to blood-letting, was coincident with the change in the prevalent opinion as to the pathology of fever, and with the substitution of relapsing fever for typhus.

The small mortality that followed the practice of blood-letting in the epidemic of 1817-19, was held up in proof of its success. The rate of mortality was contrasted with that which followed an opposite plan of treatment in the (mainly typhus) epidemic of 1800, the distinction between typhus and relapsing fever not being recognised. Welsh declared that the fever of 1817-19 was the same as had always prevailed; and that its supposed diversity 'resided in the mental revolutions of practitioners, rather than in 'the actual revolutions of disease.' But the comparative success was obviously due to the substitution of a disease which is rarely fatal, for one which is most mortal. It is now known that in relapsing fever itself, no benefit is derived from blood-letting, and even in the epidemic of 1817-19, some observers had the sagacity to discern its inutility. Dr. William Brown of Edinburgh, maintained that the cases did quite as well, which were not bled.[u] Dr. Graham of Glasgow, did not bleed for fear of typhoid symptoms, 'which would show themselves even in synocha.' The mortality in Dr. Graham's wards, was 52 in 601, or 1 in $11\frac{9}{52}$; whereas, in the wards of the other physicians to the same institution, who practised blood-letting, the deaths were 61 in 552, or 1 in 9.[v] Dr. O'Brien, of Dublin, also protested against the extent to which bleeding was practised in fever, on the mistaken notion that it depended on cerebral inflammation. His fears and predictions are not devoid of interest at the present day. 'A few years ago,' he says, 'the name—typhus fever—seemed to call for the liberal 'use of stimulants, and immense quantities of wine were accord- 'ingly given. Wine was administered indiscriminately, and, of 'course, injudiciously; it was given as well in the typhus com- 'bined with inflammation, as in its less complicated form, uncon- 'nected with visceral derangement. Wine, however, is of late 'more sparingly, and blood-lettting more frequently, employed. 'But may we not apprehend that blood-letting, the value of which

[t] Bateman, 1818, pp. 97-8. [u] Brown, 1818. [v] Graham, 1818.

'is now generally admitted, will, in its turn, be carried to excess, 'while the virtues of wine are estimated at too low a rate? Judg- 'ing of the future by the past, such an event is not impos- 'sible.'[x]

The next epidemic of fever was in 1826-28. The circumstances which ushered it in were not so much failures of the crops, as commercial distress, and hence it was confined to a few of the largest towns, and had not the wide-spread character that marked other epidemics. There had, however, been a partial failure of the potato crop in 1825. The origin of the epidemic was thus accounted for by Dr. O'Brien: 'At the conclusion of the spring 'and commencement of the summer (1826), it unfortunately hap- 'pened that a vast body of artisans resident in the Liberties of 'Dublin were thrown out of employment, and actually laboured 'under all the miseries of artificial, yet positive famine, being 'destitute of the means of purchasing food.'[y] The number of these artisans amounted to 20,000, and it is worthy of notice that the epidemic was predicted prior to its commencement. Commercial failures occurred in many other parts of the British Isles, while in Edinburgh there was a sudden failure in building speculations. The result was that the demand for labour was reduced; at the same time provisions were unusually dear. The epidemic commenced in Dublin in May, 1826, reached its acmé in October, continued stationary through the winter, and at the beginning of March, 1827, underwent a rapid and unexpected diminution. On May, 12th, 1827, the number of cases in the Cork-street Fever Hospital was reduced to 185. The number of admissions into this hospital between April 1st, 1826, and May 31st, 1827, amounted to 12,877, to which must be added the cases treated in the other Dublin hospitals and at their own homes. The number which could not be admitted into hospital was considerable, for at one time in October, 1826, it was calculated that 3,200 were ill at their own homes, and only 1,400 in all the hospitals of Dublin together. In Glasgow and in Edinburgh, the epidemic did not commence until long after its appearance in Dublin, and did not reach its acmé until 1828 (see Table I); a similar remark applies to London, where, however, the fever was much less prevalent. This epidemic, like the preceding, consisted of relapsing fever and typhus. Relapsing fever was still a prominent feature, especially in Ireland, and at the commencement of the outbreak, but true typhus was much more common than in 1817-19, and the latter part of the epidemic

[x] O'Brien, 1818, pp. 486 and 490.

[y] O'Brien, 1828, p. 515.

was made up almost exclusively of it:[z] Alison noted a measly eruption in most of the cases treated by him. Consequently, the rate of mortality was greater than in 1817-19, especially towards the close of the epidemic. Of 12,877 cases admitted into Cork-street Hospital at Dublin, between April 1st, 1826, and May 31st, 1827, 481 died, or 1 in $26\frac{3}{4}$, but of 784 cases admitted during the first three months of 1827, 47 or 1 in 16 died; and out of 1,570 cases in Edinburgh, 153, or 1 in $10\frac{1}{3}$ died. Dr. Christison, in 1857, stated that all the cases in this epidemic were treated alike by blood-letting;[a] but Alison, in an account of the epidemic, published at the time, observed, that the danger was from asthenia far more frequently than in the epidemic of 1817-19, and that wine and diffusible stimulants were 'much more frequently and 'decidedly useful in the present epidemic than formerly.' Dr. O'Brien of Dublin considered slight depletion useful in some of the relapsing cases, but maintained that, in typhus, blood-letting was wholly inadmissible, and that the best treatment consisted in wine and stimulants. Dr. Burne of London described the fever of 1827 as 'adynamic'; he pointed out that the morbid appearances found in the brain were quite independent of inflammation, that the delirium resulted from the circulation through the brain of vitiated blood, or from deficient arterial pressure, and that copious depletion protracted both the fever and convalescence, or induced a dangerous degree of debility. In reference to the profuse bleeding of former years, he observed, 'The extraordinary, I may indeed 'say, wonderful accounts, resemble more the tales of romance, and

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[z] See the remarks on this Epidemic in the Historical Sketch of Relapsing Fever.

Dr. Stokes, writing in 1854, stated that it was the so-called 'Typhoid Fever' which raged epidemically at this time. He says that disease of the intestines was the rule, and the reverse the exception, and that perforations were common. Cases of this description were undoubtedly met with, and the circumstance will be accounted for in a subsequent part of this work; but all the accounts published at the time show that the bulk of the cases were as above stated. Reid, in his account of the epidemic at Dublin, alluded to ulceration of the bowel as of occasional occurrence; but in four out of six autopsies recorded by him it is clear that the intestines were sound, and in one case only is it stated that there was 'seemingly some tendency to ulceration' (Reid, 1828). Of six cases dissected by Mr. Jacob, at Sir Patrick Dun's Hospital, the intestines were healthy in all. (O'Brien, 1828, p. 570). In Edinburgh, according to Dr. Christison, the epidemic was made up of Typhus and Relapsing Fever, and 'Enteric Typhus' did not come into notice until the end of the epidemic in 1829, and even then cases of it were very rare. (Christison, 1858, p. 588). Of twenty-six cases dissected by Alison, in not one was there ulceration of Peyer's patches (Alison, 1827, p. 258). 'Except in *autumn*,' wrote Burne of London, 'in those instances in which the attack of the adynamic fever was accompanied 'by diarrhœa or cholera morbus, there was no evidence of disease in the intes-'tinal canal' (Burne, 1828, p. 129).

[a] Christison, 1858, p. 588.

'the fiction of a sanguine imagination, than the sedate relation of 'medical facts. A medical reviewer of the day[b] stated that both in Edinburgh and in London, it was discovered in 1827 that cases of fever would not bear blood-letting. This discovery, be it observed, was made seven years before the date assigned by Dr. Christison, to the so-called change in the constitutional type of fever, and coincided with the increase in the comparative prevalence of true typhus.[c]

And now, as might have been anticipated, much difference of opinion began to prevail as to the proper treatment of Continued Fevers. Some physicians still clung to the views in which they had been brought up, and strongly advocated blood-letting,[d] while others preferred stimulants. I am informed by an eyewitness that even a few years later, Dr. Craigie, one of the physicians to the Edinburgh Infirmary, bled, cupped, and leeched his fever cases; another of the physicians poured in wine from the first; and a third did little or nothing. In connection with this it is interesting to note, that it appears from a report published by Dr. Craigie, of the fever cases under his care from 1834 to 1835, 24 died out of 174 or 1 in 7·25, while of the cases under the other physicians at the same time only 59 in 651, or 1 in 11·03, died.[e]

So completely did relapsing fever disappear from Britain after 1828, that when, after an interval of fourteen years it again showed itself as an epidemic in 1843, the junior members of the profession failed to recognise it, and it was regarded by some as a new disease. But, in the mean time, the complete substitution of the maculated typhus for the non-eruptive relapsing fever, directed especial attention to the eruptions of Continued Fever, and certain physicians fancied that they had discovered in typhus a new disease. Dr. Roupell, in a lecture before the Royal College of Physicians of London, in 1831, described typhus as a new exanthematous disease under the designation of '*Typho-rubeoloid*'[f] and

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[b] Edin. Med. & Surg. Journ. 1828, vol. xxx, p 411.

[c] For an account of this epidemic see Graves and Stokes, 1826; Alison, 1827; Burne, 1828; O'Brien, 1828; Reid, 1828; Wallace, 1828; Jacob, 1828; Stokes, 1854; and Christison, 1858.

[d] Blood-letting in fever was strongly commended by Drs. Tweedie and Southwood Smith, in their works on fever published in 1830. There were few cases, according to Tweedie, which were not benefited by blood-letting, and too often reason to regret its non-performance in the early stage; and he added, 'No remedy in the treatment of fever has been more abused than wine' (Tweedie, 1830, p. 195).

[e] Craigie, 1836. In 1837, Craigie recommended the abstraction of eighteen or thirty ounces of blood in cases of typhus, and stated that even in the advanced stage, port wine was often too strong (Craigie, 1837, No. 1).

[f] Roupell, 1831, and 1839.

Dr. Stewart states that at Glasgow previous to 1835, 'the exan-
'them of typhus, then found to be of general occurrence, had neither
'been looked for nor registered in the Infirmary, and was received
'as a new discovery.'[g] The error of such an opinion is apparent from the foregoing sketch.

After 1828, there was a considerable increase of typhus in Glasgow and Edinburgh in 1831-2, but no extensive epidemic occurred until 1836. Fever in this year became very prevalent in Ireland and afterwards in Britain (See Table I). In Dublin the epidemic commenced in 1836, reached its climax in the winter months of 1837-8, and by September 1838 had almost subsided. In 1837 alone, 11,085 cases of fever were admitted into the different hospitals of Dublin; while in Glasgow 5,387 cases were admitted into hospital, and the total number of fever cases was calculated by Dr. Cowan to be 21,800. A committee of physicians appointed to investigate the causes of the fever in Dublin, reported that they were the same as had been observed in all previous epidemics, viz.: want and overcrowding, and that these causes existed in an unusual degree. In Glasgow and Dundee, large numbers of the poor population were thrown out of employment in consequence of strikes and commercial failures, while corn and coals were unusually dear. Glasgow and Dundee were the towns in Britain that suffered first and most severely; in Edinburgh and London, the epidemic was later in making its appearance, and less extensive. The fever at this time was genuine typhus; almost every observer alluded to the appearance of the measly eruption and petechiæ. Consequently, there was a great rise in the rate of mortality over that of preceding epidemics. In 11,085 cases admitted into the Dublin hospitals, the deaths were 1,103 or 1 in $10\frac{1}{2}\frac{1}{0}$. The total deaths from fever in Glasgow in 1837 were 2,180 or 1 in 10 of those attacked; at Belfast 199 out of 1,510 cases died, or 1 in $7\frac{2}{5}$; and at St. Bartholomew's Hospital, London, 10 out of 60 died. It is true that some physicians, as Roupell and Callanan, still practised bleeding in fever; but Roupell acknowledged that the practice was much less necessary than in former epidemics. West stated that the epidemic in 'London seemed to forbid venesection,' and G. A. Kennedy found 'that at Dublin, in the great majority of instances, bleeding was 'not only inadmissible, but positively injurious.' On the other hand, wine and other stimulants were generally resorted to, and

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[g] STEWART, 1840, p. 315.

the necessity for their employment was ably advocated by Stokes.[h]

During the year 1836, typhus was also very prevalent at Philadelphia, U.S. According to Gerhard, the fever was restricted to the most crowded alleys, inhabited by the poorest of the population, and the first cases were almost exclusively from the most destitute. Bleeding was found to be injurious, while stimulants and quinine were administered liberally.[i]

The next epidemic of fever in 1843, differed from those that preceded it, inasmuch as it did not originate in, or implicate, Ireland, but was mainly confined to Scotland. There was no increase of fever in the Irish hospitals during this year (see Table I.), whereas the number of admissions into the Glasgow Infirmary rose from 1,194 to 3,467 ; in the Edinburgh Infirmary, from 842 to 2,080; and in the Aberdeen Infirmary, from 282 to 1,280. These numbers, too, are far from representing the true extent of the epidemic, for thousands of sick were sent from the hospital doors. The fever was almost exclusively relapsing fever; typhus was comparatively rare. The first cases were observed on the east coast of Fife, in 1841-2,[k] and not in the crowded localities of large towns. In Dundee, the fever appeared early in the summer of 1842, and raged to a considerable extent during the whole of the autumn, before it showed itself elsewhere. In Glasgow, the first cases occurred in September, 1842; but the fever was not generally prevalent until December, from which month the cases rapidly increased until October, 1843, when the epidemic began to decline. The number of cases in Glasgow was estimated at 33,000, or 11½ per cent. of the entire population. In Edinburgh, relapsing fever was first observed in February, 1843: it rapidly spread until October, after which it gradually abated, until, by the following April, it had well nigh disappeared. In the month of October, 1843, the number of fever cases admitted into the Edinburgh Infirmary amounted to 638, and during several months, from thirty to fifty cases were daily refused admission. The total number of cases in Edinburgh was calculated by Alison at 9000. In Aberdeen, the epidemic commenced about the same time, and followed the same course as in Edinburgh. At Leith, curiously enough, it did not appear until

[h] For the history of this epidemic, see G. A. Kennedy, 1838 ; Cowan, 1838; West, 1838 ; Graves, 1838, and 1839 ; Stokes, 1839 ; Roupell, 1839; Christison, 1858. [i] Gerhard, 1837.

[k] The fever described by Mr. H. Goodsir, as prevailing in Fife, in 1841-2, was obviously, from its symptoms, the same as what characterised the epidemic of 1842-3 (H. D. S. Goodsir, 1843).

September, 1843; it then spread rapidly for two months, after which it declined, and by the end of February, 1844, it had almost ceased; but during this brief period it attacked 1,800 persons, or one in every fourteen of the population. The disease was general over Scotland, and was not restricted to the large towns; it prevailed in Greenock, Paisley, Musselburgh, Tranent, Penicuick, Haddington, Dunbar, the Isle of Skye, etc. Although the epidemic was mostly confined to Scotland, the same fever was observed in some of the large towns of England. The number of admissions into the London Fever Hospital rose from 252, in the preceding year, to 1,385 in 1843; and the annual report for 1843 makes it evident, that a large proportion of these cases were relapsing fever. The rate of mortality of the epidemic was small, not exceeding two-and-a-half to four per cent. Although this was the same fever as prevailed in 1817-19, even local bleeding was rarely resorted to, and many of the cases were thought to demand stimulants. All accounts agree in stating, that the epidemic supervened upon a period of great distress among the Scottish poor, and that it was restricted throughout to the poorest and most wretched of the population.[1]

In 1846, commenced an epidemic of fever of unprecedented magnitude and severity, which lasted about two years. This epidemic was preceded by an extensive failure of the potato crop, which entailed an amount of wretchedness and famine, more especially among our Irish neighbours, that will not readily be forgotten. The epidemic commenced in Ireland during the last three months of 1846; at Glasgow, at the close of 1846; at Liverpool, in January, 1847; at London and Edinburgh, in March; and at Manchester, in April. It reached its climax in the summer and autumn of 1847, but did not subside until the end of 1848. There is abundant evidence to show that the fever was imported, to a great extent, by the Irish into the large towns of Scotland and England, and even to America. Apart from the circumstance that the epidemic commenced in Ireland, and first attacked those towns of Britain most accessible to Irish immigrants, it is well-known that the Irish flocked over to Britain by thousands, that in England and Scotland during the whole epidemic, the majority of persons who suffered were Irish, and that at first they were almost exclusively Irish who had but recently left their

[1] For an account of this epidemic, see references in Bibliography for 1843 and 1844; Douglas, 1845; Wardell, 1846; Christison, 1858; and Reports of the Edinburgh, Glasgow, and Aberdeen Infirmaries, and of the London Fever Hospital.

own country. During the first three months of 1847, no fewer than 119,054 Irish immigrated into Liverpool alone; and so late as June, 1847, Dr. Duncan, the officer of health, stated that the fever was entirely confined to the Irish locality, and that the health of the English inhabitants was good.[m] In Glasgow, out of 9,290 cases, 5,316, or 57 per cent, were found to be Irish, and one-third of the Irish had resided in Glasgow less than twelve months. In Edinburgh, the proportion of Irish was 73 per cent., and was particularly large at the beginning of the epidemic. Of 473 cases of fever in the Edinburgh Infirmary, on June 10th, 1847, 379 were Irish; but on July 26th, only 410 of 608 cases. At the London Fever Hospital, it was ascertained that at the outset of the epidemic, the patients were mostly Irish, who had arrived in London only a few days prior to admission, 'either with fever on them, or destitute of food and clothing, and in an extreme state of exhaustion.' In 1847, 75,000 Irish emigrated to British North America, of whom nearly 10,000 died from fever, either on the voyage, or in the Quarantine Hospitals soon after their arrival. The Quarantine Hospitals, which during the year 1847 cost the Home Government £150,000, did not prevent the fever being introduced into several towns of America. But the fever at this time was not everywhere due to Irish importation. A remarkable epidemic, similar in its nature, occurred in Upper Silesia, and other parts of Germany, where the circumstances of the population closely resembled those in which our Irish neighbours were unfortunately placed. The particulars of this epidemic will be referred to under the head of 'Relapsing Fever.'

The fever was general over Ireland. In Dublin, the lowest estimate of the number of cases was 40,000, and for the whole of Ireland, the number probably exceeded one million. In England, the total number of cases of fever in 1847, was probably upwards of 300,000. In Liverpool alone, 10,000 persons died of typhus; Manchester, Birmingham, Preston, London, and most large towns likewise suffered, although to a less extent. In Glasgow, 11,425 cases of fever were admitted into the different hospitals during 1847, in addition to the patients who were not removed from their own houses. In Edinburgh, 2,503 persons died of fever, and it was calculated that 19,254, or one in every nine of the population suffered from it.[n]

Three different fevers were observed during this epidemic. In the first place, there were a few cases of Pythogenic or Enteric

[m] See *Review*, 1848. [n] R. PATERSON, 1848, p. 386.

Fever. Most of these cases occurred at the commencement of the epidemic or before it, and were merely the remains of an extraordinary autumnal increase of this form of fever. The summer and autumn of 1846 had been remarkable for their high temperature and protracted drought, and consequently towards the end of 1846, pythogenic fever became unusually prevalent in England, *even at many places where the epidemic of typhus fever never made its appearance.*[o] It is not surprising, then, that pythogenic fever should have been unusually prevalent at Edinburgh, Glasgow, and elsewhere. Moreover, most of the Edinburgh cases occurred prior to the outbreak of the epidemic fever, and came from localities in the neighbouring country, and from the best houses of the New Town, and not from the crowded courts of the Old Town, to which the epidemic was afterwards mainly restricted.[p] The epidemic consisted essentially of Typhus and Relapsing Fever, with a preponderance of typhus in Britain, and of relapsing fever in many parts of Ireland. In the Glasgow Infirmary, where the different fevers were discriminated, the number of pythogenic cases admitted during the years 1847-8 was only 134, while that of typhus and relapsing fever was 6,225. 'In one instance only,' said Dr. H. Kennedy, of Dublin, 'did the fever so often seen in 'France, come before me.'[q]

The rate of mortality for the whole epidemic was high, but was always highest in proportion to the number of cases of true typhus. In Ireland it was only 8 per cent.; but in Edinburgh out of 19,254 cases 2,503 or 13 per cent. died; and in Glasgow out of 11,245 the mortality was 14·41 per cent. The mortality, however, of the relapsing cases alone, was in Glasgow only 6·38 per cent., and in Edinburgh 4 per cent; while that of Typhus was 21·2 per cent. in Glasgow, and 24·7 per cent. in Edinburgh.

Stimulation was the treatment almost invariably resorted to in the typhus cases; and, even in relapsing fever, depletion was seldom practised. In some places, the relapsing cases were treated successfully by stimulants. Of 179 cases of relapsing fever among Irish reapers at Croydon, treated by Mr. Bottomley with abundance of stimulants and nourishment, only four died.[r]

[o] The evidence in support of this statement will be found under the head of Pythogenic Fever.

[p] This appears from the residences of the patients given in Dr. Waters's thesis (unpublished). See *Bib.* 1847.

[q] See H. KENNEDY, 1860, *Ed. Journ.* p. 217, and *Irish Report*, 1848, viii. 56.

[r] The account of this epidemic has been obtained from most of the memoirs mentioned in the *Bibliography* for 1847, 1848, and 1849, from GRAVES, 1848, i. 97; W. T. GAIRDNER, 1859 and 1862; CHRISTISON, 1858; and from the reports of various hospitals.

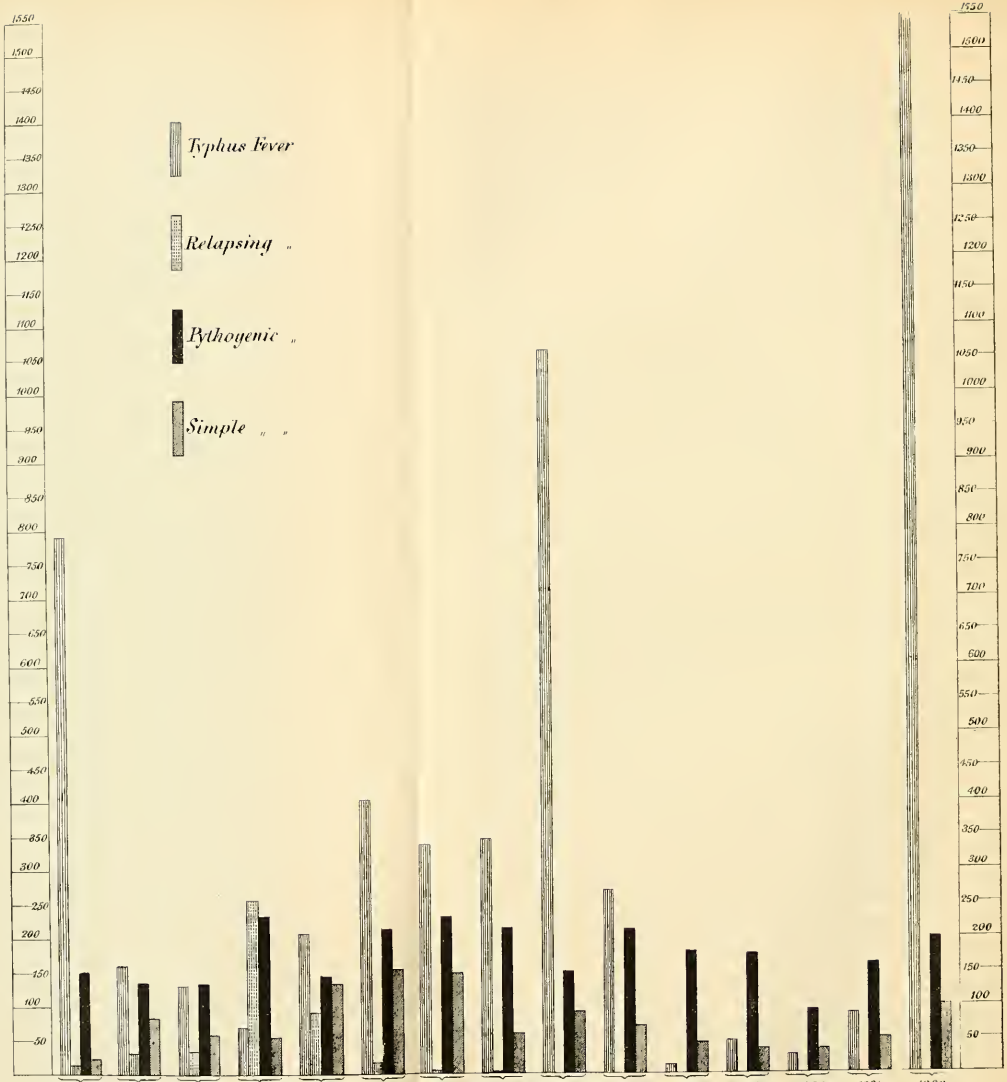

Diagram I, shows the Annual number of admissions, of each of the Continued Fevers, into the London Fever Hospital, during fifteen years.

W. West, Lith.

The last epidemic of typhus which has attracted much public attention, was that which committed such awful havoc in the French and Russian armies in the Crimea, after the capture of Sebastopol. Typhus had made its appearance during the preceding winter (1854-5) in both the English and French armies, but its prevalence was slight in comparison with that of the following winter, when it was mainly confined to the French and Russian armies. During the first six months of 1856, it was computed that out of a force of 120,000 French, 12,000 were attacked with typhus, of whom one half died. The causes of this epidemic will be considered hereafter. Pythogenic fever was also met with in the Crimean armies, and among the English was perhaps more common than typhus; but the symptoms as well as the numerous *post-mortem* examinations made by Jacquot and others, prove that the great epidemic alluded to was genuine typhus. In most of the cases, a stimulant treatment was found to be imperative.[s]

The number of typhus cases admitted into the London Fever Hospital, and the Glasgow Royal Infirmary since 1847, is given in Table II. (See also Diagram I.)

TABLE II.

Number of Cases of Typhus Fever admitted into the London Fever Hospital, and into the Glasgow Infirmary, since 1847.

Years.	London.	Glasgow.	Years.	London.	Glasgow.
1846	...	500	1854	337	760
1847	...	2399	1855	342	385
1848	786[t]	980	1856	1062	385
1849	155	342	1857	274	314
1850	130	382	1858	15	175
1851	68	919	1859	48	175
1852	204	1293	1860	25	229
1853	408	1551	1861	86	509

[s] For an account of the Fever in the Crimea, see ALFERRIEFF, 1856; BAUDENS, 1856 and 1858; LYONS and AITKEN, 1856; SCRIVE, 1857; *Review*, July 1857; ARMAND, 1858; JACQUOT, 1858; CAZALAS, 1860.

[t] With regard to the admissions for this year, it must be mentioned that the present Fever Hospital was not opened until 1849. The former building was only half the size. The precise fever was not stated in 260 cases entered in the Register for the year 1848, which was the first in which a record was kept of the different Continued Fevers in the London Fever Hospital. These 260 cases were probably mostly typhus, and hence they are included in the above 786, but not in subsequent calculations throughout this work, except when specially stated.

It will be noticed that there was a great increase of typhus in London in 1856. This increase was confined to London, and was not of Irish origin, for of 910 patients admitted into the London Fever Hospital, in regard to whom the circumstance was noted, only 53 were natives of Ireland, and all but two of the 53 had resided in London more than three months. It ensued upon a temporary distress, or artificial scarcity, among the poor. The disasters of the Crimean campaign had brought mourning into many families of the higher class, and this conjoined with increased taxation, suspense, and other causes, interrupted the ordinary gaieties of London life. Many of the working class, dependent upon the rich, were thrown out of employment, while at the same time all the necessaries of life rose greatly in price. The restoration of peace, and an abundant harvest in 1856, with increased attention to sanitary arrangements among the poor, were speedily followed by a subsidence of fever, and for four years typhus was less prevalent both in London, and throughout the United Kingdom, than at any previous period during the present century. In 1858, only fifteen cases were admitted into the London Fever Hospital, and several of them were of doubtful character; during the last six months of the year only one case was admitted. In the years 1858, 1859 and 1860, typhus was so rare a disease in London, that the students at the various hospitals had no opportunity of seeing a single case, while serious thoughts were entertained of converting the Fever Hospital into a hospital for general diseases, its mission for the treatment of typhus having, as some thought, been fulfilled.

A similar decrease took place in Scotland. Since the commencement of the present century, the number of admissions for fever into the Edinburgh and Glasgow Infirmaries, was at no time so small as during the last five or six years. In 1857, only 56 cases of typhus were admitted into the Edinburgh Royal Infirmary; in both January and May, 1858, I ascertained that the institution did not contain a single example of typhus. Writing in July, 1859,[x] Dr. W. T. Gairdner remarked on the exemption from typhus during the previous five years, and observed that more than once a considerable portion of an academic session had passed over, without his being able to show his students a single characteristic case of eruptive typhus: for several months both in 1858 and 1859, not one case was admitted into his wards. The admissions for fever into the Glasgow Royal Infirmary, in 1858 and 1859, were fewer than in any of the thirty-five preceding years, notwithstanding the enormous increase of the population during that period. But still,

[x] W. T. Gairdner, 1859, p. 241.

there was never such a complete absence of typhus in Glasgow as in London and Edinburgh. In Ireland, I am informed by Dr. Lyons, that for three or four years (1858—1861) typhus was certainly much less prevalent than formerly, although cases were by no means so rare as in Britain.

But in 1861, typhus again became epidemic. At the close of the severe winter of 1860—61, a larger number of cases were admitted into the London Fever Hospital than at any time since 1857. About the middle of December, the cases suddenly increased; and since January, 1862, the number of admissions for typhus have exceeded that at any period of the history of the hospital, while many patients have been refused admittance for want of room. From January 1st to June 30th, no fewer than 1107 cases of typhus were admitted into the Fever Hospital, and numerous cases were also under treatment in the other metropolitan hospitals. The deaths from 'typhus' returned to the Registrar-General, were more than double the ordinary average. The circumstances preceding this sudden increase did not differ from those of former epidemics. There was no failure of the crops in England, but for some time before there had been great and increasing distress among the poor of London, consequent on the organised system of strikes, the effects of which had only temporarily been averted by the relief from the societies for promoting the short-hour movement. As in 1826, 1836, and 1856, an *artificial* scarcity was the result. In addition to this, the great distress in the provinces caused the poor population of London to be *condensed* by the arrival of labourers from the country in search of work. It was ascertained that almost all the first cases admitted into the Fever Hospital were male tramps, with no fixed residence, out of employment, and suffering for many weeks from want, and that many of them had only been a few weeks in London; but there was no evidence that they had come from infected localities, or imported the fever into London. Only a small proportion of them were Irish (page 57), and none had arrived recently from Ireland. Overcrowding, with destitution, appears to have occasioned the epidemic.

In Glasgow, there has been also a considerable increase of typhus. About 800 cases were admitted into the Royal Infirmary between August 1st and December 31st, 1861, or more than five times the number admitted during the whole of the years 1858 and 1859. Here also there was no evidence that the disease was imported from Ireland.[y] As far as I have been able to ascertain, there has hitherto (Aug., 1862), been no increase of typhus elsewhere in Britain, and comparatively little in Ireland. Although there has

[y] Report of Infirmary and information from private sources.

been great distress in the cotton districts of England, the efforts to relieve it have been unparalleled, 'the pressure has not yet 'taken the form of extreme want,'[z] and there has been no evidence of unusual overcrowding of the population in the large towns.

It is remarkable, that at Edinburgh, where typhus was formerly so prevalent, only four cases were admitted into the infirmary, between November 1st, 1861, and July 29th, 1862. The non-manufacturing population of Edinburgh which did not remain exempt from typhus during seasons of *general* famine, is less readily affected by the circumstances that generate *artificial* scarcity in London and some other large towns. Yet in 1826, when Edinburgh was suffering from the effects of failures in building speculations, typhus was far more prevalent there than in London.

The foregoing historical sketch leads to the following conclusions:

1. Typhus prevails for the most part in great and wide-spread epidemics.

2. These epidemics appear during seasons of general scarcity or want, or amidst hardships and privations arising from local causes, such as warfare, commercial failures, and strikes among the labouring population.

3. During the intervals of epidemics, sporadic cases of typhus occur, particularly in Ireland, and in the large manufacturing towns of Scotland and England.

4. Although some of the great epidemics of this country have commenced in Ireland, and spread thence to Britain, appearing first in those towns on the west coast of Britain, where there was the freest intercourse with Ireland, it is wrong to imagine that all epidemics have commenced in Ireland, or that typhus is a disease essentially Irish. The disease appears wherever circumstances favourable to its development are present.

5. In many epidemics, Typhus has been associated with Relapsing Fever, and the relative proportion of the two fevers has varied greatly.

6. From the earliest times, Typhus has been regarded as a disease of debility, forbidding depletion and demanding support and stimulation.

7. The chief exception to the last proposition, originated in the erroneous doctrines taught in the early part of this century, according to which the disease was looked upon as symptomatic of inflammation or congestion of internal organs.

8. The success believed at one time to follow the practice of venesection, was only apparent. It was due to the practice having for the

[z] 'The Times,' Leading Article, Aug. 19, 1862.

most part been resorted to in cases of Relapsing Fever and acute inflammations, and to the results having been compared with those of the treatment by stimulation of the much more mortal typhus.

9. Although Typhus Fever varies in its severity and duration at different times and under different circumstances, there is no evidence of any change in its type or essential characters. The typhus of modern times is the same as that described by Fracastorius and Cardanus. The period during which epidemic fever was said to present an inflammatory type, was that in which relapsing fever was most prevalent, and the times in which the type has been described as adynamic, have been those in which relapsing fever has been scarce or absent.

Section IV.—Geographical Distribution of Typhus Fever.

THERE is probably no part of Europe in which Typhus has not been observed. Some of the greatest epidemics on record have occurred in Italy and Spain.[a] It has been described as prevailing in Germany, Belgium, Holland and Denmark, by many of the early writers[b], and more recently by Hildenbrand,[c] Hufeland,[d] Suchanek,[e] Schutz,[f] Virchow,[g] Dümmler,[h] Messemann,[i] Steensmann,[i] etc. Huss has proved its common prevalence in Sweden;[k] and numerous epidemics in various parts of Russia have been recorded by Auer,[l] Bidder,[l] Löwenstein,[l] Heimann,[l] etc. Although travellers have asserted that typhus is never seen among the Laplanders or Esquimaux,[m] it is probable from the writings of Schleisner,[n] that epidemics have often occurred in Iceland. Typhus was a common scourge of the armies under, and opposed to, the first Napoleon, in almost every country of Europe;[o] and more recently the same disease decimated the French and Russian armies in the Crimea and Turkey.[p]

It is an error to suppose that true typhus never occurs in France The works of Ambrose Paré, Fernelius, Riverius, and many other writers, prove that in early days it was a common disease there.[q] In the latter part of last century, it seems to have been not uncommon in the hospitals of Paris, and the nurses and young surgeons were often attacked by it.[r] During the first fifteen years of the present century, epidemics of typhus were very common in different parts of France; they are referred to in the works of Gaultier de

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[a] See *Historical Account*, pp. 21, 22, 23, 32.
[b] See Historical Account, pp. 23, 26. [c] Hildenbrand, 1811.
[d] Hufeland, 1814. [e] Suchanek, 1849. [f] Schutz, 1849.
[g] Virchow, 1849. [h] Dümmler, 1849. [i] Hirsch, 1859, p. 153.
[k] Huss, 1855. [l] Hirsch, 1859, p. 152. [m] Fergusson, 1846, pp. 162 & 176.
[n] Schleisner, 1850. [o] See p. 35. [p] See p. 51.
[q] See pp. 22, 24. [r] Ténon, 1788.

Claubry,[s] Jacquot,[t] Barrallier,[u] etc. Epidemics have also been observed at Beaulieu, in 1827;[x] at Toulon, in 1820, 1829, 1833, 1845, 1851, 1855, and 1856;[y] at Rheims, in 1839;[z] and at Strasbourg, in 1854.[a] Six years ago, cases of typhus were not uncommon at Marseilles, Avignon, and Paris, and other parts of France, among the soldiers returned from the Crimea.[b] It is possible also that sporadic cases of typhus occasionally occur in the large towns of France, but are mistaken for the more prevalent '*Fièvre typhoide.*' Both Andral[c] and Louis[d] confess, that in certain cases of Continued Fever, they found the intestines after death perfectly healthy; and similar observations have been recorded by Martin Solon,[e] and Piedagnel,[f] and have been reported by different observers to the French Academy. Still, such cases are unquestionably rare; while, both in France and most other parts of the continent, epidemics of typhus have of late years been observed only occasionally in large armies, or in smaller bodies of men crowded together in hulks and prisons.

It is in Britain, and still more in Ireland, that typhus has its peculiar habitat. Here, from time to time, epidemics have occurred, equalling if not surpassing in magnitude any that have been noted on the continent. And not only so; the disease, more especially in Ireland, is never absent in the intervals of great epidemics to the same extent as on the continent, but assumes more or less of an endemic character.

Although typhus is more prevalent in Ireland than in Britain, it is not imported from the former into the latter country, to the extent commonly believed.[g] The following table shows the birth-places of 3,167 typhus patients admitted into the London Fever Hospital during fourteen years (1848—1861).

TABLE III.

Natives of London	2,026	63·98	per cent.
,, rest of England	730	23·06	,,
,, Scotland	28	·88	,,
,, Ireland	350	11·02	,,
Foreigners	33	1·04	,,
Total whose birth-place was noted .	3,167	99·98	,,

[s] DE CLAUBRY, 1838. [t] JACQUOT, 1858.
[u] BARRALLIER, 1861, pp. 14 and 47. [x] HIRSCH, 1859, p. 154.
[y] KERAUDREN, 1833; FLEURY, 1833. HIRSCH, 1859, p. 154. BARRALLIER, 1861, p. 47. [z] LANDOUZY, 1842.
[a] FORGET, 1854. [b] GODÉLIER, 1856; HIRSCH, 1859, p. 154. [c] ANDRAL, 1833.
[d] LOUIS, 1841. [e] *Archiv. Gén. de Méd.* 2nd Ser. i. 400. [f] Ib. 2, vii. 410.
[g] Vide COWAN, 1858, and M'CULLOCH'S *Statistical Account of the British Empire*, 8vo Lond. 1837.

It appears, then, that only 350, or 11 per cent. of the total 3,167 typhus patients were natives of Ireland. Moreover, the majority of the 350 had been resident in London too long to have imported the disease, only 38 having been resident less than three months, and all but 63 more than a year. That typhus has been imported largely by the Irish into Britain has been admitted. It was particularly noted to be so in the epidemic of 1847–8 (see page 49); indeed, most of the 38 patients but recently arrived from Ireland were admitted into the London Fever Hospital in 1848. But of 910 typhus cases admitted in 1856, whose birth-place was noted, only 53 were natives of Ireland, and 2 only of the 53 had been resident in London less than three months, and all but three more than a year. A similar observation was made in the epidemic of 1862. Of 992 cases admitted into the London Fever Hospital, during the first six months of 1862, whose birth-place was noted, only 44 were natives of Ireland, and all but 5 of the 44 had resided in London more than three months.

But typhus in Britain has an Irish origin greater than might be inferred from the above figures, and independent of actual importation of the poison. From the census of 1851, it appears that of the 2,362,236 inhabitants of London:

2,189,883 were born in London, England, or Wales.
108,548 ,, Ireland.
30,401 ,, Scotland.
33,404 ,, other parts of the world.

Consequently there were admitted into the London Fever Hospital with typhus, in the fourteen years, 1848-61:

1 in every 310 of the Irish inhabitants of London.
1 ,, 794 ,, English ,,
1 ,, 1,086 ,, Scotch ,,
1 ,, 1,012 ,, Foreigners resident in London.

Moreover, a large proportion of the patients marked 'natives of London' were children of Irish parents or of Irish extraction. It is well-known, that by the immigration of the lower classes of Irish, pauperism and habits of overcrowding and personal uncleanliness—the main causes of the prevalence of typhus—have been greatly augmented in the large towns of Britain.

In the United States and British North America, typhus has prevailed extensively at different times, as shown by the excellent descriptions of Gerhard,[h] Bartlett,[i] and Austin Flint.[k]

There is no evidence that typhus has hitherto been observed in Australia or New Zealand.[l]

[h] GERHARD, 1837. [i] BARTLETT, 1842, 1856. [k] FLINT, 1852.
[l] HIRSCH, 1859, p. 158.

As yet, there are no authentic records of typhus, such as we see it in this country, having been met with in Asia, Africa, or the tropical parts of America. Dr. R. Dundas speaks of typhus as a common disease in Brazil; but his descriptions, and the circumstance that he found a gradual transition between the so-called typhus cases and the ordinary malarious fevers of the country, render it more than probable that the former were examples of Adynamic Remittent Fever.[m] Dr. Ewart[n] has described two cases of 'typhus,' as occurring in the jail of Ajmere in Bengal; but the characteristic eruption was absent, and there was no evidence of contagion. Dr. Allan Webb, in his '*Pathologia Indica*,' had previously mentioned two cases of fever observed at Simlah, where petechiæ were present.[o] But the fever is not said to have been contagious, and petechiæ are occasionally observed in the severe remittents of India, which have often been mistaken for typhus. According to Dr. Morehead, typhus is unknown on the continent of India.[p] Accounts have been published of typhus occurring in Mexico, Central America, and South America,[q] but none of the descriptions, which have come under my notice, make it conclusive that the disease was true typhus, and not the ordinary typhoid or adynamic remittent fever of these countries.

In 1861, Dr. W. Walker of H. M. Indian army, described an epidemic of 'typhus' prevalent in the preceding year in the north-western provinces of India, and observed by him in the central prison of Agra.[r] But the evidence that the disease was genuine typhus, as seen in this country, is to my mind not conclusive. An eruption was never detected upon the skin, although it was carefully looked for; the swarthy skin of an East Indian would not suffice to obscure the eruption of typhus in its petechial stage; for I have known the eruption distinctly developed in Africans and East Indians. Without insisting on the difficulty of diagnosing typhus, when no eruption is present, it may be remarked that the disease differed from true typhus in several respects. 1.—In 23 out of 104 cases, there was jaundice, a symptom of such extreme rarity in typhus that Dr. Jenner never observed it.[s] 2.—The duration was much shorter than the ordinary duration of typhus; of 229 fatal cases, 9 died within twenty-four hours, 105 within five days, and 176 within ten days. 3.—Relapses were very frequent on the third or fourth day of convalescence, while

[m] DUNDAS, 1852. [n] EWART, 1856.
[o] *Pathologia Indica*, Lond. 1848, p. 212.
[p] *Clinic Res. on Dis. of India*, 1st ed., i. 307.
[q] HIRSCH, 1859, p. 157; DUNDAS, 1852. [r] WALKER, 1861.
[s] JENNER, 1853.

in typhus, as we shall find, relapses are so rare, that most observers have doubted their occurrence. 4.—The disease, at its commencement, presented a remittent type. 5.—Neither the patient nor his attendants could tell exactly at what time the fever left him, a character directly opposed to what is constantly noticed in typhus. The disease differed from remittent fever and resembled typhus in its apparently contagious character, although the evidence on this head is not crucial. A large proportion of the attendants upon the sick caught the disease, but no instance is mentioned of its importation by a tainted person into a previously healthy district.

It is important to notice, that attention has of late years been directed to the occurrence in different parts of India of an 'Adynamic Remittent Fever of suspected infectious character,' better known by the designations *Pali Disease* and *Mahamurree*. For an excellent summary of what has been written on this disease, the reader may refer to the second edition of Dr. Morehead's 'Clinical Researches on Disease in India.'[t] It may be here stated, that the disease is believed to be contagious, that it is remittent in character, but with great tendency to become continued, and that adynamic phenomena are well marked. In none of the cases have petechiæ, or a measly eruption been observed; but in the great majority, glandular swellings of the groin, axillæ, and neck, have been present from the first. The mortality has been great: according to one observer, four-fifths of those attacked perished. This disease closely resembles, if it be not identical with, bubonic plague. Like both the plague and typhus fever, it 'has prevailed chiefly amongst the poor, in filthy, badly 'ventilated houses, and villages; and has been preceded by seasons 'of famine.'.

And here I may anticipate an opinion subsequently expressed, to the effect that there exists a strong analogy, if not identity, between typhus fever and true plague, the poisons being generated from similar causes, and differing only in intensity from the effects of climate and other collateral circumstances. Plague is probably the typhus of warm climates. There are few subjects more deserving of investigation than that of contagious fever in the tropics. Dr. Morehead thinks it not improbable that remittent fever may assume adynamic or typhoid characters, and at the same time become infectious, in consequence of overcrowding and neglect; and this may have been the real explanation of the epidemic described by Dr. Walker. It is not unreasonable to suppose

[t] 2nd Ed., London, 8vo., 1860, p. 155.

that, under such circumstances, the fever is the result of malaria with a poison resembling that of typhus superadded. The etiological relations of typhus and "yellow fever," are also well worthy of investigation.

It is important to know, that the natives of tropical countries are often attacked by typhus on visiting localities where it is prevalent. I have known several Africans and East Indians admitted with typhus into the London Fever Hospital, the rash being distinct. Gerhard states, that in the Philadelphia epidemic of 1836, the majority of persons attacked were negroes or mulattoes.[x]

Section IV.—Etiology of Typhus Fever.

THE causes of Typhus are the Exciting and Predisposing. The primary exciting cause is a specific poison. The properties of this poison, the question whether it be ever generated *de novo*, or always derived from an infected person, must engage our attention. Under predisposing causes, those circumstances will be referred to, which in themselves are insufficient to generate the disease, but which predispose the body to the influence of the primary exciting cause, and without which, the latter would often prove inert. Some of these predisposing causes may be called determining causes, from their determining, so to speak, the action of the poison.

A.—Predisposing Causes of Typhus.

1.—*Sex.*

Sex in itself does not predispose to Typhus. That both sexes are equally liable appears from the following table, which gives the sex of the typhus patients admitted into the London Fever Hospital, during fourteen years.

Table IV.

Years . .	1848	1849	1850	1851	1852	1853	1854	1855	1856	1857	1858	1859	1860	1861	Total.
Males . .	290	88	59	31	135	211	177	161	450	135	7	14	14	53	1824
Females	236	67	71	37	96	197	160	181	612	139	8	34	11	34	1856

Thus out of 3,680 cases of Typhus, the females only exceeded the males by 32; while in one-half of the fourteen years, the number of males was greater, in one year (1852) almost double that of the females.

From an examination of statistics, extending over a limited period, it might be inferred that typhus attacked one sex in preference to the other; but the fact of its doing so is owing to accidental circumstances. In the recent outbreak in London, the

[x] Gerhard, 1837, xix. 296.

cases were at first exclusively males, nineteen out of twenty-one admitted in December, 1861, belonging to this sex; but the men were tramps who were out of work, and many of whom had but recently arrived in London, without wives or families. As the epidemic advanced, the females exceeded the males. The preponderance of male cases, in the Scotch epidemic of 1847, probably admitted of a similar explanation. In 1847, 1,419 males and 980 females, suffering from typhus were admitted into the Glasgow Royal Infirmary,[y] while in the Edinburgh Infirmary there were 3,677 males to 2,226 females.[z]

The statement that sex in itself has no influence over the prevalence of typhus, holds good in regard to the other continued fevers, although opinions of an opposite nature have often been expressed. Indeed, the conclusions of different writers on this point are contradictory. Thus, Dr. Harty, in his historical sketch of the fever epidemic in Ireland, in 1817-19, found that there were 34,398 females to 32,144 males.[a] Dr. G. A. Kennedy ascertained that in the Dublin hospitals, in the year 1837, there were 6,099 females to 4,086 males;[b] and Dr. Orr showed, that of the cases admitted into the Glasgow Fever Hospital during fifteen years (1831-45), there were 16,834 females, to 15,863 males.[c] On the other hand, of the cases of fever treated in the Edinburgh Infirmary during eleven years (1839—1849), there were 10,811 males to 8,863 females;[d] and of 3,186 admitted into the Seraphim Hospital at Stockholm, 2,181 were males.[e] These varying results at different times and places, are no doubt owing to a preponderance of one sex in the population, or to local and accidental circumstances, which expose one sex more than the other to the exciting causes of fever, or which influence the admission into hospital of one sex in preference to the other. Including all the forms of Continued Fever admitted into the London Fever Hospital during fourteen years, there has been a remarkable equality of the sexes, 3,780 being males, and 3,792 females.

2.—*Age.*

Typhus is, for the most part, a disease of adult age, although no period of life is exempt from it. The mean age of 3,456 cases admitted into the London Fever Hospital during ten years, I have

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[y] Steel, 1848, p. 161.

[z] *Statist. Tables*, 7th Ser. p. 11. These figures included a considerable number of cases of Relapsing Fever; but the proportion for typhus only was similar, for of 1,069 typhus cases under Dr. R. Paterson (*Bib.* 1848) and Dr. W. Robertson (*Bib.* 1848) there were 588 males to 481 females.

[a] Harty, 1820. [b] G. A. Kennedy, 1838, p. 43.

[c] Orr, 1846, p. 345. [d] *Statist. Tables*, 10th Ser., p. 20. [e] Huss, 1855, p. 42.

ascertained to be 29·33 years, which is about four years above the mean age of the total population.[f] The following Table gives the number of cases of typhus admitted into the London Fever Hospital, in each quinquennial period of life during ten years, 1848-57 (see Table V. and Diagram II.)

TABLE V.[g]

Age.	No. of Cases.			Per centage at each period of life.
	M.	F.	M. & F.	
Under 5 years	9	8	17	·49
From 5 to 10 years ...	88	95	183	5·29
,, 10 to 15 ,, ..	175	188	363	10·47
,, 15 to 20 ,, ...	295	251	546	15·79
,, 20 to 25 ,, ...	287	208	495	14·32
,, 25 to 30 ,, ...	185	158	343	9·92
,, 30 to 35 ,, ..	157	166	323	9·34
,, 35 to 40 ,, ...	121	149	270	7·81
,, 40 to 45 ,, ...	109	183	292	8·44
,, 45 to 50 ,, . .	98	114	212	6·13
,, 50 to 55 ,, ...	71	79	150	4·34
,, 55 to 60 ,, ...	48	52	100	2·89
,, 60 to 65 ,, ...	40	48	88	2·54
,, 65 to 70 ,, ...	17	25	42	1·21
,, 70 to 75 ,, ...	9	15	24	·69
,, 75 to 80 ,, ...	4	2	6	·17
Above 80 years	1	1	2	·06
Age doubtful	23	27	50	—
Total, omitting doubtful cases	1714	1742	3456	99·90

From this table it appears, that the two most common lustra for typhus have been between fifteen and twenty, and twenty and twenty-five, and that one half of the cases (1707) occurred between fifteen and twenty-five. Moreover, nearly one half (43·66 per cent.) of the cases were thirty or upwards, and almost one-eighth (11·9 per cent.) were fifty or upwards; while less than one-sixth (16·3 per cent.) were under fifteen. Two circumstances also must be

[f] The mean age of the total male population of England and Wales was for 1821, 25·13; for 1841, 25·49; and for 1851, 25·87 (*Reports of Census of* 1851.)

[g] In this Table, a patient who had completed his fifth or tenth year was reckoned as being between 5 and 10, and 10 and 15 years respectively; and so on, for all the other periods of life.

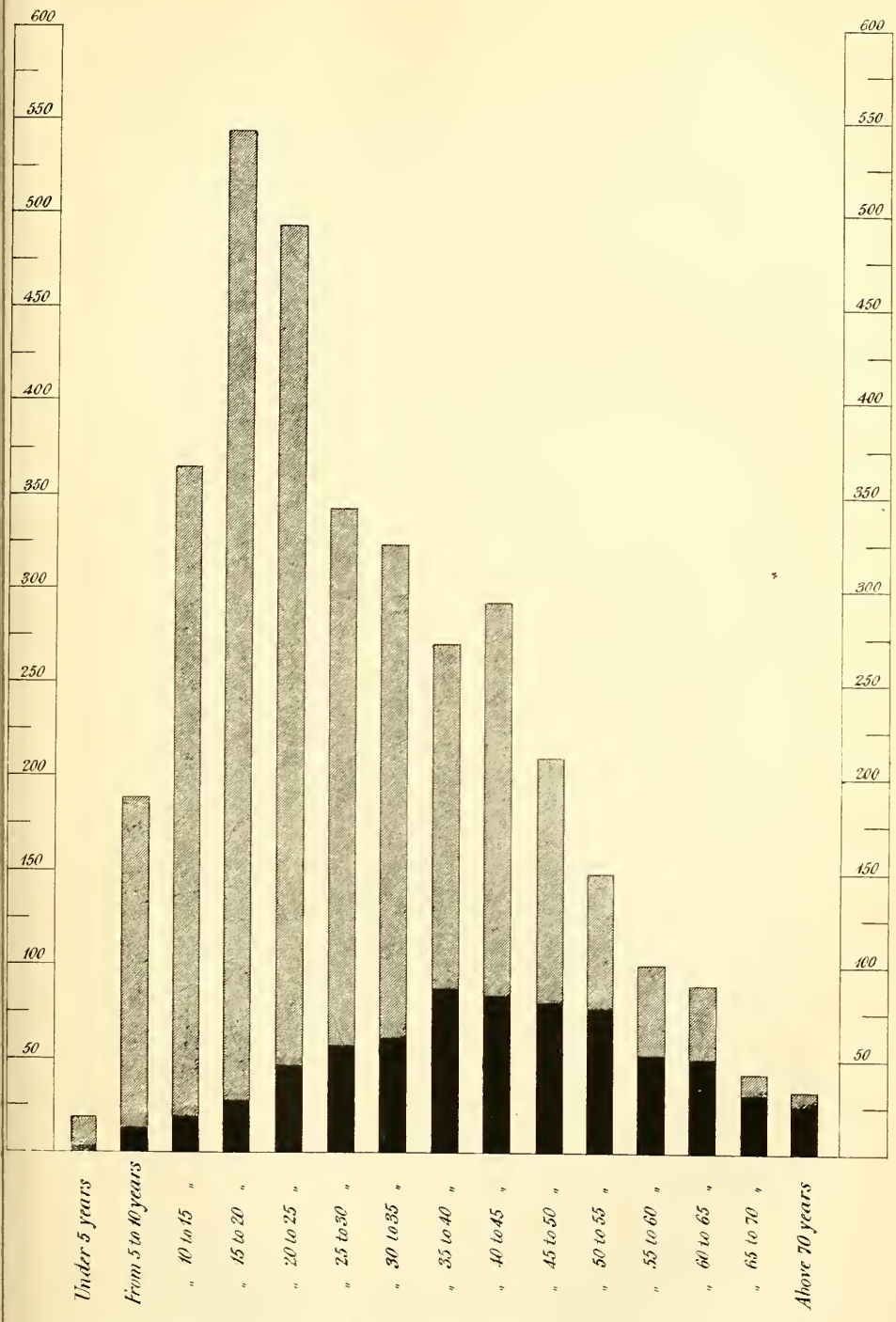

Diagram II, shows the ages of 3456 cases of Typhus Fever, admitted into the London Fever Hospital, with the number of deaths ■ *at each age.*

borne in mind, which indicate that the liability to typhus after 30, is much greater than it appears to be from the Table. In the first place, the total number of the population above 30 years of age is scarcely more than one-third of that below;[h] and secondly, in many persons above 30, the liability to typhus is perhaps removed by the fact of their having already had the disease. The youngest case of typhus observed in the London Fever Hospital during the period in question was an infant under twelve months; the oldest, a man aged eighty-four.

Females of an advanced age would appear to be more prone to typhus than males. Thus in every one of the ten years, the mean age of the female typhus patients exceeded that of the males, and taking the ten years collectively, the mean age of 1742 female cases was 30·27; that of 1714 males only 28·38. Again

Out of a total of 3,456 cases		1,714	were males, and	1,742 females
Of those below 15 years		272	,,	291
,,	below 30 ,,	1,039	,,	908
,,	above 30 ,,	675	,,	834
,,	above 40 ,,	397	,,	519
,,	above 50 ,,	190	,,	222

Another circumstance worthy of notice is, that the number of females admitted with typhus between the ages of 40 and 45, instead of being less, as in males, is absolutely greater, than in any of the three preceding lustra. The changes which the female system undergoes at this period of life seem to predispose to attacks of typhus.

The conclusions arrived at under this head confirm, on a great scale, most previous observations, the chief exception being the greater susceptibility to typhus among females of an advanced age than among males. Dr. Cowan found the contrary result at Glasgow in the epidemic fever of 1836.[i]

Dr. Peacock[k] has shown that the ratio per cent. of typhus to the general admissions into the Edinburgh Infirmary, for the year ending September 30th, 1842, was greatest under fifteen years of age, and diminished progressively with the advance of life; but young children are rarely admitted into hospital for general diseases, while of those affected with typhus, a disproportionate number are sent to hospital owing to the contagious nature of the

[h] According to the census of 1851, the total population of England and Wales amounted to 18,109,410 persons, of whom, 13,771,056 were under thirty years of age, and 4,338,354 over thirty.

[i] Vide Cowan, 1838. [k] Peacock, 1843, p. 7.

malady, and to the circumstance of whole families being often struck down by it at once.

The fact that adult age is so prone to typhus involves social and moral consequences of the highest importance. This disease attacks and destroys the heads of families, at that period of life when they have children dependent upon their industry for support. Hence it is often one of the main causes of widowhood and orphanage, and therefore of pauperism and demoralisation.

3.—*Months and Seasons of the Year.*

Table VI, and Diagrams III & IV, show the number of cases of typhus admitted into the London Fever Hospital, during the months, quarters, and seasons of fourteen successive years.

Taking the fourteen years collectively, May, April, and March, were the months in which there was the greatest number of admissions; September and December those in which there was the smallest; the largest number was in spring, the smallest in autumn. But this distribution was far from constant in the different years. Thus in 1853, the number of cases in spring only exceeded that of autumn by one, and in 1850 and 1855 there were most cases in autumn, and fewest in spring. Again, in 1851, there were 5 cases in summer and 22 in winter, while in 1854, there were 155 cases in the former season, and 45 in the latter, and in 1859 there were 20 cases in summer and only 2 in winter. At Glasgow, in 1845, the largest number of cases occurred in January; but in 1847, in July.

Epidemics of typhus thus appear to commence and progress irrespectively of season, so long as other known causes of the disease continue in operation. This conclusion is confirmed by a careful comparison of the most authentic records of various epidemics. At the same time, typhus is often observed to be most prevalent in the latter half of winter, in the spring and beginning of summer, and many epidemics have declined rapidly towards the end of summer. Taking all the fourteen years comprehended in the above Table, the largest number of cases occurred in May, and from this month the numbers progressively diminished to one-half in September. The Glasgow epidemic of 1847 was at its height in July, and afterwards gradually declined. (See Table VI.).

Turning to those periods when typhus is no longer epidemic, the few cases met with occurred mostly in spring, and in autumn the disease entirely disappeared. Thus from April 26th, 1858, to March 12th, 1859, only two cases of typhus, with the

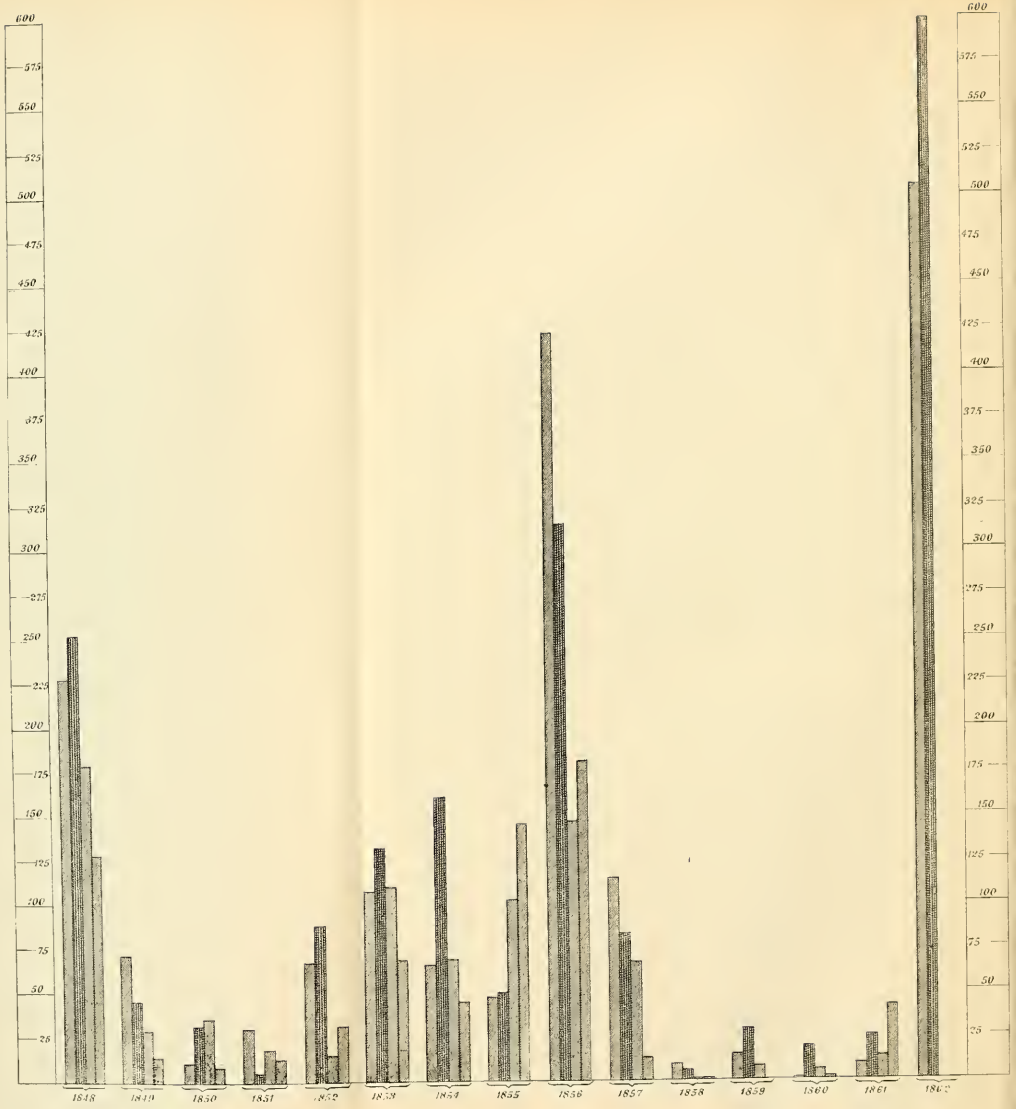

Diagram III shows the Quarterly admissions of Typhus Fever, into the London Fever Hospital, during fifteen years. (The second quarter is shaded darker, to contrast with Diag. VIII.

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Table VI.

Typhus Fever. Months and Seasons.[1]

	1848[m]	1849	1850	1851	1852	1853	1854	1855	1856	1857	1858	1859	1860	1861	Total.	1862	Glasgow Infirmary.	
																	1845.[n]	1847.[o]
Jan. .	43	27	2	11	30	31	11	19	157	54	3	2	—	2	392	141	38	66
Feb. .	37	19	5	11	8	26	15	20	124	35	3	—	1	2	306	154	29	93
March.	66	25	4	8	28	47	38	9	140	24	2	11	—	4	406	210	29	88
April .	65	14	9	3	41	47	38	6	134	23	2	13	1	11	407	224	20	135
May .	66	17	9	—	34	42	73	17	96	42	—	10	5	10	421	215	24	218
June .	43	13	13	3	15	43	50	27	87	18	3	6	11	4	336	163	16	226
July .	48	7	22	2	13	23	32	30	68	35	1	4	1	4	290		29	400
Aug. .	32	9	20	7	2	51	21	42	39	16	—	1	4	6	250		12	246
Sep. .	39	11	16	9	4	32	13	29	39	14	—	1	—	2	209		17	235
Oct. .	22	6	13	3	6	36	12	51	55	10	—	—	1	12	227		11	227
Nov. .	44	4	9	7	8	13	17	38	70	1	—	—	1	8	220		18	262
Dec. .	21	3	8	4	15	17	17	54	53	2	1	—	—	21	216		23	203
Spring.	168	58	18	22	77	120	91	35	398	82	4	24	2	17	1116		78	316
Summer	157	37	44	5	62	108	155	74	251	95	4	20	17	18	1047		69	844
Autumn	93	26	49	19	12	119	46	122	133	40	—	2	5	20	686		40	708
Winter	108	34	19	22	53	61	45	111	280	57	7	2	1	31	831		79	531
Total	526	155	130	68	204	408	337	342	1062	274	15	48	25	86	3680		266	2399

[1] Under 'Winter' are included January, November, and December of the same year.

[m] The 260 cases referred to in note, page 5, are omitted from the Table, but included in the diagram. They were distributed through the twelve months of the year as follows: 24, 29, 29; 27, 34, 16; 17, 26, 18; 5, 19, 16.

[n] Orr, 1846. [o] Steele, 1848.

characteristic eruption were admitted into the London Fever Hospital, one on December 16th, the other on January 25th; while from enquiries made at the time, it was ascertained that no cases were admitted into the other London hospitals during that period. In the four months, however, March, April, May and June 1859, as many as 40 cases of typhus were admitted into the Fever Hospital. In the nine following months, only 9 cases were admitted; but again in April, May, and June 1860, 17 cases. Lastly, during the eight months succeeding June 1860, only 11 cases were admitted, but in March, April, May, and June 1861, 29 cases.

It is to be noted, that typhus does not always become more prevalent with the commencement of the cold weather; neither does it immediately decline on the advent of summer. A considerable duration of cold weather appears to be necessary before it increases, and the greater prevalence thus induced, does not cease until after a protracted duration of warm weather, while an epidemic is often at its height in the middle of summer. Hence the frequent increase of typhus in the latter half of winter and in spring is not referrible to mere cold, but is more probably owing to the protracted overcrowding and more defective ventilation of the dwellings of the poor during the cold weather. This view of the matter is confirmed by what was observed in the French army in the Crimea. Here are Jacquot's remarks :—'Pas de typhus l'été, 'alors que le soldat vit en plein air et laisse ouvertes les baraques 'ou les tentes. Avec la saison rigoureuse, le typhus se développe 'deux fois de suite, et deux fois de suite il se dissipe au retour 'de la saison chaude, qui permet la ventilation des demeures et la 'vie à l'air libre.'[p]

4.—*Temperature and Moisture.*

From what precedes, it is obvious that the ordinary variations of temperature, in this climate, have little influence over the prevalence of typhus. In Glasgow, the epidemic of 1847 was at its height in July,[q] whereas in the same city, ten years before, typhus was most prevalent during six weeks of hard frost, when the ground was covered with snow.[r] The hygrometric states of the atmosphere have likewise no effect on the prevalence of typhus. The influence of different climates is discussed under the head of Geographical Distribution.

It is not unusual for patients to attribute the disease to their having 'caught cold' or 'got wet.' Thus, of 1828 Typhus

[p] JACQUOT, 1858, p. 64. [q] See Table (page 65). [r] PERRY, 1844, p. 84.

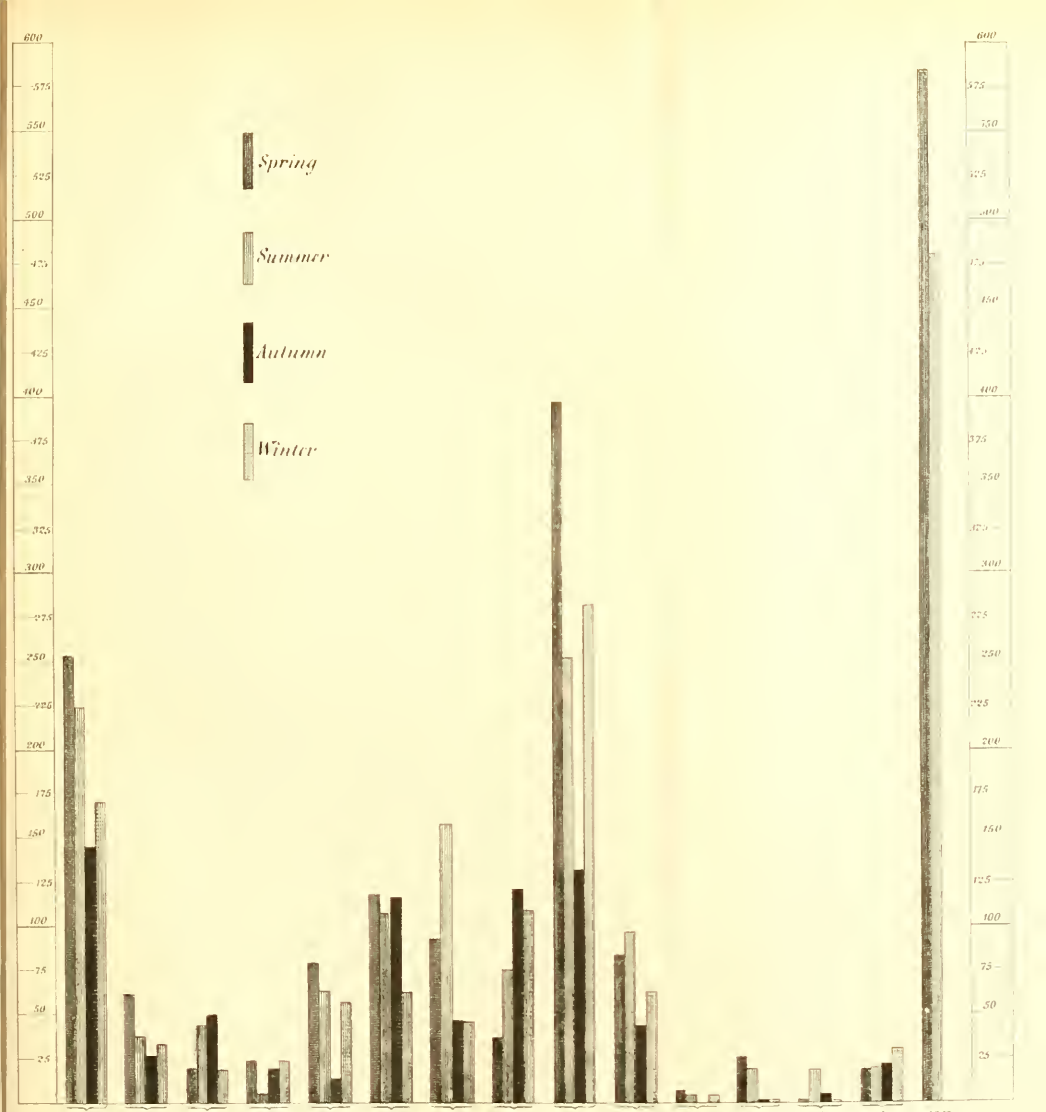

Diagram IV shows the number of admissions of Typhus Fever into the London Fever Hospital, during each Season of fifteen years. (Compare with Diag. IX.)

patients treated in the London Fever Hospital during seven years, 123, or 9·78 per cent. blamed one or both of these causes for their illness. Exposure to cold and wet, especially if long continued, independently of its exciting catarrh or local inflammation, has a depressing influence on the nervous system, and so favours the advent of typhus. Occasionally the poison of typhus seems to be stored up for some time in the system, and does not take effect until after some such exposure, which then constitutes a "Determining Exciting cause," and is often mistaken for the exciting cause itself.

5.—*Occupation.*

Table VII. shows the occupation of 5,095 fever patients admitted into the London Fever Hospital during ten years (1848—1857). In 2713 of the cases, the fever was typhus. (see p. 68).

No occupations in themselves predispose to typhus, except those involving actual exposure to the specific poison. A large proportion of the patients admitted into the London Fever Hospital are out-door labourers; and many, although belonging to some trade, have been out of employment for weeks or months prior to their seizure. Butchers are said by Dr. Tweedie[s] to be particularly exempt from typhus: the statement is probably correct, and the fact is accounted for by the circumstance that butchers have usually a good supply of nourishing food. Most of the 18 butchers noted in the Table had been out of employment and destitute for some time before their illness.

6.—*Individual Idiosyncrasy.*

Individual idiosyncrasy may predispose to typhus. According to Armand,[t] many of the French soldiers in the Crimea, appeared to enjoy perfect immunity from the disease, although placed in circumstances identical with those of others who contracted it. It is difficult to say to what extent other predisposing causes may have operated. During the present epidemic (1862), one of the resident medical officers of the London Fever Hospital has been in close communication daily for eight months with numerous cases of typhus, but has hitherto escaped, although every nurse and attendant not fortified by a previous attack, has suffered. On the other hand, there are persons who seem to have a peculiar aptitude for contracting typhus, and who, in the course of their lives, have more than one attack.

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[s] Tweedie, 1830, p. 79. References, however, to the occurrence of typhus among butchers will be found in Smith, 1830, p. 431; Mateer, 1836, p. 38; Craigie, 1837, (2), 289–91; G. A. Kennedy, 1838, p. 37; Peacock, 1843.

[t] Armand, 1858, p. 409.

Table VII.

		Typhus.		Relapsing.		Typhoid.		Febricula.	
	Total.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Female Servants...	958	321	11·82	38	12·42	461	31·64	138	22·29
Male ditto.....	36	10	·36	...	...	20	1·37	6	·96
Artizans[1].....	662	380	13·1	23	7·51	186	12·76	73	11·79
Shoemakers.....	204	138	5·08	10	3·26	41	2·81	15	2·42
Tailors.....	62	37	1·36	6	1·96	11	·75	8	1·29
Sempstresses	372	242	8·92	10	3·26	91	6·24	22	4·68
Bakers	39	12	·44	1	·32	20	1·37	6	·96
Householders	14	10	·36	...	...	4	·27	...	...
Shopmen	145	60	2·21	6	1·96	67	4·59	12	1·93
Schoolmasters	10	8	·29	...	...	2	·13	...	...
Scholars	90	31	1·14	2	·65	40	2·74	17	2·74
Clerks	32	11	·40	...	...	17	1·16	4	·64
Butchers	33	18	·66	1	·32	11	·75	3	·48
Barmen	32	12	·44	2	·65	14	·96	4	·64
Policemen	45	10	·36	...	...	30	2·05	5	·8
Soldiers andSailors	51	22	·81	6	1·96	10	·68	13	2·1
Milk-men and women	21	12	·44	1	·32	6	·4	2	·32
Travellers.....	4	3	·11	...	...	1	·06	...	...
Porters and Errand Boys.....	146	63	2·32	13	4·24	52	3·57	18	2·9
Hawkers and Street Musicians	247	136	5·01	54	17·64	24	1·64	33	5·32
Shoeblacks	7	4	·14	...	...	1	·06	2	·32
Cabmen & Ostlers	97	53	1·95	3	·98	35	2·4	6	·96
Cowkeepers	10	1	·03	...	...	9	·61	...	...
Labourers[2]	837	436	16·07	87	28·43	184	12·62	130	21·
Dustmen	12	9	·33	1	·32	...	...	2	·32
Sweeps	6	4	·14	1	·32	1	·06	...	...
Charwomen	171	125	4·61	9	2 94	16	1·09	21	3·34
Laundresses	208	154	5·67	9	2·94	31	2·12	14	2·22
Vagrants.....	64	44	1·62	12	3·92	...	...	8	1·29
" Paupers "[3]	106	83	3·05	3	·98	8	·54	12	1·93
Nurses in Workhouses	54	46	1·79	...	...	1	·06	7	1·11
Bridewell and Ho. of Correction ..	14	13	·47	...	...	...	...	1	·16
Nurses, London Fever Hospital..	40	25	·92	1	·32	3	·20	11	1·77
Married Females[4].	266	180	7·73	7	2·28	60	4·11	19	3·02
Total of which occupation known.	5095	2713	100.	306	99·9	1457	99·82	619	99·7

[1] These include all in-door workers, except those otherwise specified, such as smiths, carpenters, printers, &c.

[2] These include all out-door workers, such as masons, dock-labourers, gardeners, &c.

[3] These have been entered into the Register as "paupers," Many who were really paupers have been entered under the occupations which they previously followed.

[4] Many other married females have been entered as following some occupation.

7.—*Intemperance.*

Habitual intemperance deranges digestion, impairs nutrition, prevents the proper oxygenation of the blood, causes degeneration of the animal tissues, and lowers the tone of the nervous system. It is not surprising, that under such circumstances, the body becomes more susceptible of the poison of typhus. It was shown by Craigie[u] and Davidson,[x] that more than one-half of the patients admitted with typhus into the Edinburgh and Glasgow Infirmaries had led intemperate lives.

A single act of intemperance may also predispose to typhus, persons who would otherwise escape. I have known several instances of persons exposed for many months to the specific poison in its most concentrated form, who were not attacked until immediately after a debauch. There is no greater error, than to imagine that a very liberal allowance of alcoholic stimulants fortifies the system against contagious diseases.

8.—*Bodily Fatigue.*

Fatigue, want of sleep, or whatever lowers the vital energies and exhausts and debilitates the body, predisposes to typhus. Instances are constantly occurring of medical students and hospital clerks, who contract the disease under the influence of such predisposing causes. It is also probable that the state of sleep favours the advent of typhus, owing to the nervous depression and languid circulation accompanying this condition. The attendants upon typhus patients ought not to sleep in the same room.

9.—*Mental Fatigue and Depressing Emotions.*

Mental fatigue and the depressing passions have an undoubted influence in rendering the body less able to resist the poison of typhus; whilst hope, confidence, and the other exciting emotions have a contrary effect. Of the predisposing causes included under this head, perhaps none is more powerful than a dread of the disease. Some years ago, a remarkable illustration of this fact made a deep impression upon me. A medical student at Edinburgh had such a dread of typhus, that he could scarcely be induced to enter a ward in which there were any cases; yet he was one of the first of the students who fell a victim to the disease in the epidemic of 1847. Depressing passions are often one of the many predisposing causes of typhus in armies and prisons.

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[u] Craigie, 1837 (2). p. 296. [x] Davidson, 1841, p. 64.

10.—*Previous Illnesses.*

Previous ailments predispose to typhus. A person often escapes the contagion of typhus for a long time, but he contracts a febrile catarrh, or an attack of simple fever, and then he falls a prey to the poison. In hospital practice, convalescents from other diseases are often attacked by typhus. Scurvy is generally admitted to be a powerful predisposing cause of typhus: it was found to be so in the epidemic of 1847-8, and in the French army in the Crimea.[y]

According to Hildenbrand,[z] typhus rarely attacks persons labouring under phthisis. Out of several hundreds of cases of typhus which came under his notice, not one was phthisical. Of 100 typhus cases dissected by Davidson, traces of tubercle were found in the lungs of only 3.[a] I am inclined to doubt the correctness of Hildenbrand's opinion. Tubercle in the lungs is far from being a rare complication or sequela of typhus, and in most such cases there is a history of phthisis prior to the attack of fever. Jenner[b] records the case of a phthisical child, who was attacked by typhus and died from a rapid deposition of fresh tubercles in the lungs; and several cases of the same nature have come under my notice.

11.—*Recent Residence in an Infected Locality.*

The following Table shows the length of residence in London of all the typhus patients admitted into the London Fever Hospital during fourteen years, with regard to whom the point was noted:—

Table VIII.

Less than 3 months	120	or	3·87 per cent.
„ 6 „	160	„	5·16 „
„ 1 year	213	„	6·87 „
„ 2 years	271	„	8·74 „
„ 10 „	557	„	17·96 „
More than 10 years, but not for entire life	518	„	16·71 „
For entire life	2,026	„	65·33 „
Total	3,101	„	100·00 „

Thus of 3,101 cases, only one-fourteenth had resided in London less than a year, and only one twenty-sixth less than three months; while 65⅓ per cent. had resided in London all their lives, and 82 per cent. more than ten years. (See page 57).

It has been long known, that the poison of Enteric Fever,

[y] Jacquot, 1858, p. 77. Barrallier, 1861, p. 38. Lind believed scurvy to be a preservative against typhus, and a similar opinion has more recently been expressed by Boudin and Dalmas.

[z] Hildenbrand, 1811, p. 144. [a] Davidson, 1841. [b] Jenner, 1850, xx, 457.

operates more readily on persons who have but recently been subjected to its influence, than on those who are habituated to it: this character does not apply to typhus. The nurses and attendants on the sick of typhus, acquire no immunity from mere exposure, unless they have already had the disease; and there is no evidence to prove, that when typhus appears in a house, it selects the new comers by preference.

In some epidemics, however, a considerable proportion of the persons attacked have but recently arrived in the infected locality; but then they have either brought the disease with them, or they are predisposed to it by their destitute condition; or they propagate, or perhaps generate, the poison by causing overcrowding.

12.—*Over-crowding.*

Over-crowding of human beings with deficient ventilation is one of the most powerful predisposing causes of typhus. Admitting that typhus is propagated by emanations from the sick, it is obvious, that its propagation must be favoured by the concentration of these emanations. All the historians of the great epidemics of typhus testify to the intimate connection between its prevalence and over-crowding.

The following Table (IX.) constructed from the register of the London Fever Hospital, shows the localities of the metropolis from which 6,520 cases of fever were derived during ten years, as well as the area and population of each district.[a] The returns of no hospital could be better suited for the purpose, as the patients are brought from every district of the metropolis. It is true, that, from various circumstances, some districts send a larger proportion of their fever cases to the hospital than others, so that the returns do not correctly indicate the *amount* of fever in each district, yet they furnish a fair criterion of the *form* of fever prevalent in each.

It will be noticed, that the typhus cases have come for the most part from the central and most crowded districts, and that on approaching more suburban districts their proportion gradually diminishes. Unfortunately, no district of the metropolis is entirely exempt from over-crowding, otherwise the contrast would be more striking. In Edinburgh, where there is a greater separation between the over-crowded dwellings of the poor and the houses of the better class than perhaps in any other city, typhus, even in the midst of the greatest epidemics, is almost restricted to the most crowded

[a] The districts are those into which London has been divided under the "Metropolitan Local Management Act."

Table IX.

	Divisions and Districts of London.	Area in Statute Acres.	Population in 1851.	Total Fever Cases from each district.	Number and per centage of each Fever in each District.							
					Typhus.		Relapsing.		Pythogenic.		Febricula.	
					No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
I. (*a.*)	Kensington . . .	1,942	44,053	74	53	71·62	—	—	16	21·92	5	6·75
„ (*b.*)	Paddington . . .	1,277	46,305	14	1	7·14	—	—	12	85·71	1	7·14
„ (*c.*)	Fulham . . .	4,155	29,646	29	14	48·27	—	—	8	27·58	7	24·13
II.	Chelsea . . .	865	56,538	26	9	34·61	1	3·84	10	38·46	6	23·07
III. (*a.*)	St. George's, Hanover Sq.	581	33,196	9	1	11·11	—	—	7	77·77	1	11·11
„ (*b.*)	Belgravia . . .	580	40,034	6	1	16·66	—	—	4	66·66	1	16·66
IV.	Westminster . .	917	65,609	119	92	77·31	—	—	21	17·64	6	5·04
V.	St. Martin's in Fields .	305	24,640	180	104	57·77	7	3·88	27	15·	42	23·33
VI.	St. James's, Westminster	164	36,406	103	53	51·45	1	·97	35	33·98	14	13·59
	West Division .	10,786	376,427	560	328	58·57	9	1·61	140	25·	83	14·82
VII.	Marylebone . .	1,509	157,696	257	152	59·14	5	1·94	77	29·96	23	8·95
VIII.	Hampstead . .	2,252	11,986	16	4	25·	—	—	10	62·5	2	12·5
IX.	St. Pancras . .	2,716	166,956	503	275	54·67	11	2·18	165	32·8	52	10·33
X.	Islington . . .	3,127	95,329	596	237	39·76	16	2·68	252	42·28	91	15·26
XI.	Hackney . . .	3,929	58,429	83	20	24·09	2	2·41	53	63·85	8	9·63
	North Division .	13,533	490,396	1455	688	47,28	34	2·33	557	38·28	176	12·09
XII.	St. Giles's . . .	245	54,214	19	8	42·1	—	—	9	47·36	2	10·52
XIII.	Strand . . .	174	44,460	293	195	66·55	9	3·07	53	18·09	36	12·28
XIV.	Holborn . . .	196	46,621	781	443	56·72	165	21·12	91	11·65	82	10·5
XV.	Clerkenwell . .	380	64,778	365	222	60·82	9	2·47	95	26·02	39	10·69
XVI.	St. Luke . . .	220	54,055	140	116	82·85	—	—	17	12·15	7	5·
XVII. to XIX.	City of London . .	723	129,128	744	406	54·57	102	13·71	133	17·87	103	13·84
	Central Division .	1,938	393,256	2342	1390	59·35	285	12·17	398	17·	269	11·49

XXI.	Bethnal Green . .	760	90,193	30	12	40·	2	6·66	15	50·	1	3·33
XXII.	Whitechapel . .	406	79,759	44	16	36·36	17	38·63	8	18·18	3	6·82
XXIII.	St. George's in the East.	243	48,376	541	343	63·4	29	5·36	100	18·48	69	12·75
XXIV. (*a*)	Limehouse . . .	576	54,173	9	2	22·22	—	—	7	77·77	—	—
„ (*b*)	Mile-End . . .	681	56,602	74	56	75·67	—	—	14	18·92	4	5·4
XXV. (*a*)	Poplar . . .	1,490	28,384	1	—	—	—	—	1	100·	—	—
„ (*b*)	Bow	1,428	18,778	6	—	—	—	—	5	83·33	1	16·66
	East Division . .	6,230	485,522	738	433	59·67	48	6·5	178	24·12	79	10·7
XXVI.	St. Saviour . . .	250	35,731	22	18	81·81	—	—	4	18·18	—	—
XXVII.	St. Olave . . .	169	19,375	50	31	62·	4	8·	7	14·	8	16·
XXVIII.	Bermondsey . .	688	48,128	64	43	67·18	1	1·56	15	23·44	5	7·81
XXIX.	St. George, Southwark	282	51,824	34	27	79·41	3	8·82	2	5·88	2	5·88
XXX.	Newington . . .	624	64,816	199	80	40·2	21	10·55	51	25·62	47	23·61
XXXI.	Lambeth . . .	4,015	139,325	593	284	47·89	9	1·51	190	32·04	110	18·55
XXXII (*a*., *b*.)	Clapham and Wandsworth . . .	3,711	25,901	186	67	36·02	6	3·22	83	44·62	30	16·12
„ (*c*)	Battersea . . .	2,343	10,560	24	3	12·5	—	—	17	70·83	4	16·66
„ (*d*)	Putney . . .	2,176	5,280	3	1	33·33	—	—	2	66·66	—	—
„ (*e*)	Streatham . . .	—	—	6	1	16·66	—	—	5	83·33	—	—
„ (*f*)	Tooting . . .	561	2,122	1	1	100·	—	—	—	—	—	—
XXXIII.	Camberwell. . .	4,342	54,667	177	62	35·02	18	10·17	75	42·37	22	12·43
XXXIV.	Rotherhithe . .	886	17,805	4	—	—	—	—	2	50·	2	50·
XXXV (*a*)	Greenwich . . .	3,771	66,998	9	4	44·44	—	—	4	44·44	1	11·11
„ (*b*)	Woolwich . . .	1,596	32,367	2	1	50·	—	—	1	50·	—	—
XXXVI.	Lewisham . . .	17,224	34,835	—	—	—	—	—	—	—	—	—
	South Division . .	45,542	616,635	1374		45·34	62	4·51	458	33·33	231	16·81
	Beyond London Districts	—	—	51	8	15·7	—	—	36	70·6	7	13·73
	Doubtful . . .	—	—	108	36	33·33	3	2·77	53	49·07	16	14·81
	Total . . .	78,029	2,362,236	6628	3506	52·89	441	6·55	1820	27·46	861	12·99

and wretched parts of the Old Town. Again, in the country districts of England, typhus is a rare disease; almost all the examples of 'typhus,' reported as occurring in small country towns and villages, are really cases of pythogenic fever.

13.—*Destitution and Starvation.*

Destitution and deficient alimentation are the most powerful predisposing causes of typhus.

The influence of poverty on the prevalence of typhus is borne out by the experience of the London Fever Hospital. On investigating the condition in life of 3,492 typhus patients admitted during ten years, it was ascertained that they belonged almost invariably to the lowest classes of the population, 94 per cent. being the inmates of workhouses or dependent on parochial relief, whereas comparatively few of the better class of patients, such as gentlemen's servants and persons able to pay for admission, were affected with typhus.[h] And not only has this been so, but it has been constantly found that a large proportion of the typhus patients have been on the very verge of starvation, for several weeks or months prior to admission.[i]

Indeed, in London, typhus is almost unknown among the middle and upper classes, save in a few isolated instances where there has been direct intercourse with the sick. I am informed by Drs. Tweedie and Jenner that they have scarcely ever met with an instance of typhus among the better classes, except in the case of medical practitioners and students, and my own experience confirms this statement. It is true, that persons even in the highest ranks are constantly said to die of typhus, but the term 'typhus' is so commonly employed to designate any form of fever, or indeed any disease with typhoid symptoms, that no weight can be attached to such statements.

From the historical account of typhus, it appears that all the great epidemics, which have devastated Ireland, Great Britain, and other parts of the world, have occurred during seasons of scarcity and want. In some instances, the famine has been general, owing to failures of the crops, and the epidemics have been wide-spread; while in others, the scarcity has been the result of artificial causes, such as strikes, commercial failures, sieges, &c., and the epidemics have been circumscribed. But, whatever has been the cause of the scarcity, it has been a common observation in many epidemics that

[h] For the precise numbers, see section on *Etiology of Pythogenic Fever.*

[i] See pages 49, 53.

the fever has raged among the poor in a degree proportionate to the privations they have endured.[k] It was so in the epidemic of 1817-19,[l] and in 1847 it was found at Dublin that those persons who had been reduced by insufficient food were first attacked, while, in many instances, the fever first showed itself on recovery from the primary effects of famine.[m] The same observation was made at Philadelphia in 1836.[n]

The influence of destitution in propagating epidemic fevers (typhus and relapsing) was long since insisted on by Bateman, who observed: 'Deficiency of nutriment is the principal source of 'epidemic fever;'[o] while in later times it was almost proved to demonstration by Alison,[p] who even believed that 'the exist-'ence of epidemic fever is a most important test to the legis-'lator of the destitute condition of the poor.' The same views were supported, although carried too far, by Dr. Corrigan[q] of Dublin, in a pamphlet published in 1846, entitled 'Famine and Fever, as Cause and Effect in Ireland.' Dr. Corrigan's memoir elicited, within a few months, an able essay from the pen of Dr. Henry Kennedy,[r] which requires some notice. Dr. Kennedy endeavoured to show, that epidemic fever was independent of famine, and that there was evidence to make it probable that, under certain circumstances, an excessive use of food might help to generate it. A reply to the more important of Dr. Kennedy's arguments, will be found under one or other of the following heads:—

1. Some of Dr. Kennedy's arguments were fallacious, from his having confounded typhus with pythogenic or enteric fever. Outbreaks of the latter fever, which is independent of destitution, and which is met with among rich and poor alike, cannot legitimately be adduced as evidence in disproof of the influence of destitution on the spread of typhus. It is well-known that in 1846, prior to the last great epidemic, a fever was prevalent not only in Ireland, Scotland, and the large towns of England, where typhus afterwards raged so fiercely, but also in many country districts of England, which entirely escaped the subsequent epidemic. Dr. Kennedy alluded to this fever as prevailing in the autumn of 1846, in Berkshire and London, to show that epidemics of fever might commence among the well-fed. This fever, however,

[k] Alison, 1840, No. 1, p. 22.
[l] Barker and Cheyne, 1821.
[m] *Irish Report*, 1848.
[n] Gerhard, 1837, xix. p. 297.
[o] Bateman, 1818, pp. 4 and 11.
[p] Alison, 1840, Nos. 1 and 2.
[q] Corrigan, 1846.
[r] H. Kennedy, 1847.

was not typhus, but Pythogenic Fever.[s] The outbreak of pythogenic fever at this time in Edinburgh, described by Bennett[t] and Waters,[u] was of peculiar importance, as under ordinary circumstances the disease was not common there. (See page 50). Although few Irish physicians distinguished the different forms of Continued Fever, the following extract from Dr. Popham's Report[x] of the epidemic at Cork, is to the point: 'The state of health in this 'city was not below the average during the early part of 1846. 'Fever of a *gastric* type was rather prevalent in May, but no 'serious amount of illness existed before the failure of the potato 'crop.' The very hot summer of 1846, preceded a failure of the crops, but seasons remarkable for a high temperature are characterised by an increased prevalence of pythogenic fever, whether the crops fail or not.

2. Dr. Kennedy stated that in certain epidemics, and particularly those of 1740, 1817, and 1836, there was an increase of sickness or fever, before the commencement of famine. But to admit this argument, it would be necessary to know more of the amount and nature of the sickness or fever alluded to, and of the precise condition of the population, than is perhaps now possible. If the argument be just, it is difficult to understand how the able observers who saw, and wrote on these epidemics, attributed them to an unusual amount of privation among the poor (see pp. 29, 34, 46).

3. It was urged by Dr. Kennedy, that epidemics of fever have been observed to continue *after* food has become plentiful. But to say nothing of the persistence of numerous foci of contagion, it is not surprising, that persons whose constitutions have been enfeebled by long want should remain predisposed for some time after plenty is restored. Indeed, some observers have thought that, during an epidemic of typhus, a sudden change from a deficient and unwholesome diet, to a full supply of nutritious food, renders the body more susceptible.[y] This is the only way in which a superabundance of food can contribute to the spread of epidemic fever. Still, I believe it is a fact, that most epidemics have declined soon after the restoration of plenty.

4. It was stated by Dr. Kennedy, that the epidemic of 1826-7,

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[s] On this point, see section on the *Predisposing Causes of Pythogenic Fever*. 'We assert with confidence,' says a writer in the *British and Foreign Medical Review* for April 1848 (p. 287), 'that the excess of fever in the 'autumn of 1846 did not constitute the *foyer* from which sprang the fearful 'irruption of 1847'.

[t] BENNETT, 1847. [u] WATERS, 1847. [x] *Irish Report*, 1848, viii. p. 278.

[y] GRAVES, 1848, i. 96.

actually subsided in Dublin, while the wants of the population were as great as when it commenced. The statement may be true ; but, before admitting it as a proof that the prevalence of typhus is not influenced by destitution and famine, it is necessary to consider certain peculiarities of the epidemic in question. It was not preceded by a general famine from extensive failure of the crops, but it was due to local, or to use the expression of one of the historians of the epidemic, *artificial* scarcity. Twenty thousand artizans in Dublin were thrown out of employment in the spring of 1826, and were actually starving. These 20,000, then, with their wives and families, included all who were *unusually* predisposed, and when they all had contracted fever, the material, so to speak, for the epidemic was exhausted. Now it was shown that, within twelve months, the number of persons attacked far exceeded 20,000 (see p. 43).[z]

5. It was argued that epidemics of fever might occur without any famine; and the argument is just, if, according to Dr. Kennedy, typhus and pythogenic fever be regarded as one disease. The epidemic of 1771, however, recorded by Sims,[a] and alluded to by Dr. Kennedy, was probably typhus; but it does not appear to have been very extensive, and the accounts of it are certainly too meagre to warrant the statement that it was not preceded by unusual privation. Although Sims made no mention of famine, he stated that the fever prevailed principally among the poor, and among those of the middle ranks, who led irregular and intemperate lives.

6. Lastly, Dr. Kennedy appealed to the circumstance, that notwithstanding the failure of the crops, the year 1846 in Ireland had been 'unusually healthy and free from fever.' But he wrote on the eve of one of the greatest Irish epidemics of typhus on record.

A careful study of the records of typhus epidemics, demonstrates, in my opinion, the intimate connection between these epidemics and famine or distress. They have appeared during every variety of climate, season, and weather: famine and over-crowding have been the sole conditions common to them all. In fact, on more than one occasion, epidemics of typhus have been predicted from the occurrence of famine; and the result has verified the prediction.

Some persons imagine that famine from failure of the crops, and epidemics of typhus, both result from one common cause, such as an obscure 'atmospheric,' or 'epidemic influence.' But against

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[z] Reid, 1828. [a] Sims, 1773.

such a view it may be argued, first, that in bodies of men living in the same locality, and exposed to the same atmospheric influences, the prevalence of typhus has been found to be in a direct ratio to the degree of privation. Contrast, for example, the condition of the English and French armies in the Crimea in 1855 and 1856. At the commencement of the siege, the English commissariat was inferior to the French, and the English suffered most from typhus. But in 1856, says Jacquot, "Le temps 's'écoule; les rôles changent.' 'L'insuffisance, et surtout la mau-'vaise qualité, des vivres de l'armée Française en Crimée sont un 'fait notoire, et déjà historique.' 'Aussi, les nouvelles épidémies 'et de scorbut et de typhus, continuant à sevir en proportion de 'l'état des armées, n'ont elles aucune prise sur les Anglais, aux-'quels rien ne manque en fait de bien-être, tandis qu'elles affai-'blissent et déciment l'armée Française.'[c] Secondly, epidemics of typhus appear during the state of privation consequent on strikes, commercial failures, and warfare; or, in other words, artificially induced famine entails the same results, as the famine arising from failure of the crops.

But it is not contended that famine can produce typhus, nor would it be right to say with Corrigan, 'If there be no famine, 'there will be no fever.' The circumstances, which are believed to generate the typhus poison, although they often coexist with famine and destitution, are quite distinct. What is here maintained is; that destitution is the chief predisposing cause of typhus, that it predisposes the constitution to the action of the specific poison at times when the latter would otherwise be inert, and that in this way famine causes a rapid diffusion of the fever, and converts a few isolated cases into a general epidemic. Moreover, famine and destitution from want of work have the effect of concentrating the poor in the large towns, and so producing over-crowding, from which the disease originates. Famine only *generates* typhus, in so far as it causes over-crowding.

B. Exciting Causes of Typhus.

The primary exciting cause of typhus is a specific poison emanating from the bodies of persons previously infected (contagion), or generated *de novo*. The contagious character of typhus has been attested by most observers since the time of Fracastorius. From this property, indeed, many of its appellations have been derived (See *Synonyms*, p.17.) Charles Maclean,[d] however, in an

[c] Jacquot, 1858, pp. 85, 92. [d] Maclean, 1817, i. 119.

elaborate work on the plague, published in 1817, strongly opposed the notion that any epidemic diseases could be communicated by contagion; but his arguments are a melancholy example of facts misinterpreted in the light of preconceived opinions. Lassis[e] also, and other writers have denied that typhus is contagious. Even at the present day, some difference of opinion exists on the point. Some eminent physicians maintain that typhus always results from contagion, and that the specific poison is never generated *de novo*;[f] others regard it as doubtfully contagious, although this conclusion is usually arrived at from observation of pythogenic fever and not of true typhus; while some sanitary reformers go so far as to assert, that there is no such thing as contagion, and that the so-called 'contagious diseases' result, in every instance, from inattention to sanitary arrangements. It is essential, that the profession and the public should have clear and decided views on this matter; and it is therefore purposed to devote a few pages to a consideration of the more important arguments and facts in favour of the contagious character of typhus, of the laws by which its specific poison appears to be governed, and of the question whether this poison always emanates from a person previously infected, or may not under certain conditions be generated independently.

1. *Contagion.*[g]

The belief that typhus is contagious, is based on such facts as the following:—

A. *When typhus commences in a house or district, it often spreads with great rapidity.* It is not uncommon for an entire family, or all the residents in a large lodging-house, to be attacked in succession. Thus, on July 2nd, 1857, seven members of one family were admitted into the London Fever Hospital in different stages of well-marked typhus, and often, ten, twenty, thirty, or even one hundred cases follow one another in rapid succession, in the same house or court. Of 2,811 cases of typhus admitted into the London Fever Hospital during eight years, at least 729 or 28·13 per cent. referred the origin of the disease to contagion. But the mere circumstance of many persons being successively attacked with typhus in the same house or district, is not a conclusive proof that the disease spreads by contagion, because the fact may be explained on the supposition of some local cause. Other proofs, therefore, are required.

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[e] LASSIS, 1819. [f] WATSON, 1848, ii. 761; W. BUDD, 1861.

[g] Here and elsewhere in this work, the word "contagion" is used in its widest signification, and not to imply actual contact.

B. *The prevalence of typhus in single houses or in circumscribed districts, is in direct proportion to the degree of intercourse between the healthy and the sick.* In a common lodging-house, it is the persons living in the same room with the first case, who are first attacked. Again, in hospital practice, the nurses and attendants on the sick rarely escape. In 1814, typhus was introduced by some soldiers into the Salpétrière at Paris; 120 persons attached to the hospital were attacked, and 8 physicians died.[h] The following facts are recorded in reference to the great Irish epidemic of 1817-19.[i] In the Cork Fever Hospital, 198 cases of fever occurred within eighteen months among the attendants on the sick. 'No clinical clerk, apothe-'cary, unseasoned nurse or servant escaped.' In the Dublin Fever Hospital, 13 of 47 attendants on the sick took fever in the course of eight months. In Steven's Hospital, 'none of the 'nurses, none of the porters, barbers, or those occupied in the 'handling, washing, and tending on the sick, escaped.'[k] In the Edinburgh Infirmary, during the year 1827, ten clinical clerks and twenty-five nurses or servants caught typhus. All of them had frequent and close communication with the fever patients; whereas the clerks and nurses, residing in the same building, who had no intercourse with fever patients, almost uniformly escaped.[l] In 1837, Dr. Cowan thus wrote concerning the typhus then prevailing in Glasgow:—'All the gentlemen 'who have acted as clerks in the Fever Hospital for many 'years past have been attacked with fever, unless they had it 'previous to their election. During the last year, twenty-seven 'of the nurses of the establishment were seized, and five of 'them died.'[m] Similar testimony is borne by West[n] and Roupell[o] with regard to typhus in St. Bartholomew's Hospital in 1837-8. Dr. W. T. Gairdner writes concerning the epidemic of 1847-8, in Edinburgh, as follows:—'In no single instance known to me 'did a nurse (in the infirmary), who had not had fever previously, 'remain for six weeks attached to a fever ward, without catching 'the disease. So much was this danger known at the time, that 'in the end no nurse was ever appointed to a fever ward, unless 'she had passed through the disease; and even, with this precau-'tion, many were infected. During the whole course of the epi-'demic, 22 resident medical officers were engaged in the fever-

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[h] R. Williams, 1836. [i] Barker & Cheyne, 1821; Harty, 1820, p. 151.

[k] In many of these cases, the disease communicated was no doubt relapsing fever, of which this epidemic was mainly composed.

[l] Alison, 1827, p. 238. [m] Cowan, 1838, p. 26.

[n] West, 1838, p. 143. [o] Roupell, 1839, p. 52.

'wards, of these 3 had previously had fever; 12 were seized 'when on duty in the hospital; and of these, 3 died. There 'were also 9 physicians who, without being resident, served 'in fever-wards; of these, 6 had previously passed through 'fever; the other three were all seized, two with typhus, and one 'with relapsing fever, and of the two cases of typhus one died.' Moreover, of the resident medical officers at this time, who escaped fever, some served exclusively in the surgical department, while others were comparatively little exposed.[p]

During fourteen and a half years, (1848-62), 80 cases of Typhus originated in the London Fever Hospital. Thus:—

Of the Nurses	44	took Typhus.
„ Medical Attendants	5	„ „
„ Servants	3	„ „
Patients admitted with Enteric Fever	10	„ „
„ „ Relapsing „	1	„ „
„ „ Febricula „	6	„ „
„ „ Scarlet „	9	„ „
„ „ Other Diseases	2	„ „

The average number of medical attendants and nurses is about eighteen. Of the servants in the establishment not engaged in the wards, only 3 had typhus, and one of them had to receive and wash the patients on admission.

Remarkable illustrations of the contagious character of typhus were furnished by the Crimean campaign. The following will suffice:—An official return showed that during two and a-half months of 1856, no fewer than 600 of the attendants in the French Hospitals at Constantinople were seized with typhus, which was not prevalent in the town itself.[q]

c. *Persons in comfortable circumstances and living in localities where the disease is unknown, are attacked, on visiting infected persons at a distance.* Although typhus rarely occurs in the middle and upper classes, living in large well-ventilated houses, members of the medical profession and the clergy, who visit the sick, have been too frequently its victims. During the Irish epidemic of 1817-19, about 40 physicians took the disease in the province of Munster, and in the single county of Kerry, 10 Roman Catholic and 3 Protestant clergymen were reputed to have died of it.[r]

Some startling facts of this nature have been published by

[p] W. T. Gairdner, 1862, No. i. 359. [q] Jacquot, 1858, pp. 95, 100.
[r] Harty, 1820, p. 151.

Drs. Stokes and Cusack. During twenty-five years previous to 1843, out of 1220 practitioners in charge of 406 medical institutions in Ireland, 560 suffered from 'typhus fever'; 28 of them twice, and 9 three times. Of the 1220, 300 died; and of the 300 deaths, 132, or nearly three in seven, were from typhus. Again, from March 25th, 1843, to January 1848, there died of the medical profession in Ireland 443; and of the 443 deaths 199 were from typhus. In the year 1847 alone, it was calculated that no fewer than 500 medical men in Ireland, or about one-fifth of the total number (2650), suffered from typhus, of whom 127 died.[s]

In Edinburgh, Dr. Christison states that, during a period of thirty-two years, he and two of his colleagues had attended upwards of 280 medical students for fever caught in the infirmary or fever hospital.[t]

I have myself had two attacks of typhus with the characteristic eruption. Once in 1847, while living in a part of Edinburgh, where there was no fever, I contracted it in the prosecution of my studies. Ten years later, while residing in a district of London enjoying complete immunity from typhus, I was again attacked, in consequence of visiting the Fever Hospital. I am not aware that one of the many hundred medical men living in the same part of London ever had typhus, except after similar exposure.

D. *Typhus is often imported by infected persons into localities previously free from it.* Many such instances are recorded by Lind with regard to the vessels of the fleet, in the last century.[u] It was often found, that the disease first showed itself on board a ship, immediately after some of the crew had had communication with another ship, already infected. The epidemic at Carlisle, in 1782, is another illustration. The disease was traced to a single house, from which it was communicated by one of the residents to several of his fellow-workmen in a distant part of the town, whence it spread to the rest of the inhabitants.[x]

The three following illustrations are on the authority of Dr. Alison.

Queensberry House had existed in Edinburgh for a century, was long occupied as a private residence by the noble family of that name, and was afterwards tenanted by a number of families in succession. During all this time, there was no record of its being the seat of fever. The building, however, was converted

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[s] Stokes and Cusack, 1848. [t] Christison, 1850, p. 267. [u] Lind, 1763.
[x] Heysham, 1782.

into a hospital, and fever cases were admitted, whereupon the resident physician and every nurse took fever in succession.[y]

The son of a shoemaker in Edinburgh lay ill of typhus, in the same house where his father and two apprentices were at work. Two or three weeks after, both of the apprentices were laid up with fever at their own houses, one 200 yards, the other a mile and a half, distant from the workshop. There immediately followed a succession of cases among the other inhabitants of the same, and of the immediately adjoining rooms, who had never been at the workshop. In one of these houses, seven, and in the other twelve persons, were thus attacked. Moreover, both of these houses were situated in localities, which for years before had been perfectly free from fever.[z]

In 1826, a labourer, his wife, and four children were attacked with typhus at Edinburgh. The father and two sons were taken to the infirmary, while the mother and two other children, being ejected from their dwelling during convalescence, took refuge in the house of a friend living in another part of the town. When the father and sons left the infirmary, the whole family removed to a third house, at a considerable distance from either of the former. There had been no fever in any of the three places they had thus successively inhabited; yet many of the inhabitants of the same story in which they first lived (and no others in that neighbourhood) had fever immediately after them; in the little court to which the mother and two children next removed, thirty cases of fever occurred within a few weeks after, the inhabitants of the same room being first attacked. In the third lodging-house, four cases of fever occurred within a fortnight after their arrival.[a]

Dr. Roupell relates that, in the spring of 1831, typhus broke out among the lower classes of sea-faring men inhabiting the north bank of the Thames. They were sent in boat-loads to the Seamen's Hospital Ship. The disorder imported into the ship soon spread among the attendants and patients admitted for other diseases. Seven extra nurses were employed to attend on the fever-patients, who, when off duty, returned to their homes on the south bank of the river, where no fever was prevalent. Six of the seven nurses were attacked with the fever, which spread in their families.[b]

Lastly, it often happens, in the general hospitals of London and other towns, that not a single case of typhus originates in the wards for many years, when the admission of one or two cases is

[y] Alison, 1827, p. 238. [z] Ibid. [a] Ibid. [b] Roupell, 1839, p. 53.

followed by a rapid spread of the disease among the inmates. This was the case in several of the metropolitan hospitals at the commencement of the present year (1862).

E. *The contagious nature of typhus is indicated by the success attending the measures taken to prevent its propagation, more especially the early removal of the sick.* Evidence in support of this proposition will be found in the works of John Clark,[c] Stanger,[d] Bateman,[e] Harty,[f] and Alison.[g] The last writer observes: 'We 'should have little difficulty in pointing out above a hundred 'houses, where a single case of fever has occurred, where the 'patient has been speedily removed, and where there has been no 'recurrence. But we should hardly find five houses in all the 'closes of the old town, in which a patient in fever has lain during 'the whole or even half the disease, and in which other cases have 'not speedily shown themselves."

With such evidence as the foregoing, few will deny that typhus is contagious. Let us now consider, so far as we know them, the laws by which the specific poison is governed.

1. *Manner in which the typhus-poison is transmitted from the sick to the healthy.* Actual contact with the sick is unnecessary for the transmission of the disease. There is every reason to believe that the poison is contained in the cutaneous and pulmonary exhalations of the sick, that it is conveyed through the air or by fomites, that it is then inhaled, or perhaps swallowed with the saliva, and so finds admission into the blood of the healthy. Every physician who has had any experience of typhus, must be familiar with the strong odour of the breath, and still more with that which escapes on turning down the bed-clothes of the patient. It has been found that those patients are most apt to communicate the disease, in whom this odour is strongest;[h] and there are numberless instances of persons being attacked with typhus, a few hours, or immediately, after close communication with a typhus patient, during which they had been strongly impressed with this pungent odour.[i]

2. *The distance that the typhus-poison can be transmitted through the atmosphere.* Haygarth, in 1777, was the first to devote attention to this question, and from extensive observation he concluded, that in the open air, 'the infectious distance of small pox does 'not exceed half a yard,' and that the contagion of typhus is

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[c] CLARK, 1802. [d] STANGER, 1802. [e] BATEMAN. 1818. [f] HARTY, 1820.
[g] ALISON, 1827, p. 312. [h] GERHARD, 1837. [i] MARSH, 1827.

'confined to a much narrower sphere.' He also observed, 'When 'the chamber of a patient ill of an infectious fever is spacious, 'airy, and clean, few or none of the most intimate attendants will 'catch the distemper. Among the middle and higher ranks of 'society in Chester and its neighbourhood, during a period of 'thirty-one years, I scarcely recollect a single instance of the 'typhus fever being communicated to a second person, not even 'during the epidemics of 1783 and 1786.'[j] Before the above was written, a remarkable observation, tending to the same conclusion was made by Lind. A large number of Spanish prisoners were confined in Forton prison in 1780. Typhus broke out among them with great severity. During seventeen weeks, 785 cases were admitted into hospital, of whom 156 died. At the same time, 229 Americans were confined in another part of the same prison: they were not allowed any intercourse with the other prisoners; but the hospital containing the sick Spaniards ranged along one side of their airing ground, and had near the ceiling of each ward ventilators opening towards the airing ground. Not one of the Americans was attacked with fever.[k] Haygarth's opinion has been confirmed by all subsequent observations. There never was an instance of typhus spreading from the London Fever Hospital to neighbouring houses, even when the hospital was one of a row of houses in Gray's-inn Lane.[l] Dr. Christison, speaking of the medical students who had contracted typhus at the Edinburgh Infirmary, and who had been attended at their own homes by himself and two of his colleagues during thirty-two years, remarks: 'I am 'sure I am within the limit when I say, that we three have 'attended 280 cases of this kind, that 1,200 persons must have 'been more or less exposed in attending on them, and only one 'instance of communication is known to have occurred.'[m] How very different is Scarlet Fever in this respect!

The late Dr. Gregory published an observation, which he believed to furnish an undeniable proof that typhus fever may spread in private houses of the better class. In a house near Cavendish Square, several cases of fever occurred in rapid succession, a servant who had recently arrived being the first to suffer. The symptoms are not given; but several circumstances render it probable, that the disease was pythogenic fever, and that it depended on some local cause.[n]

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[j] Haygarth, 1801, pp. 8, 9, 38, 39.
[k] See Clark, 1802, p. 23.
[l] R. Williams, 1836, i. 38.
[m] Christison, 1850, p. 267.
[n] Gregory, 1832, p. 746.

From all experience, it follows, that if a typhus patient be placed in a large, well ventilated apartment, the attendants incur little risk, and the other residents in the same house, none whatever. There are likewise no grounds for the popular belief, that typhus may be propagated through the atmosphere from a fever hospital to the houses in its neighbourhood. On the other hand, medical attendants who auscultate typhus patients, or who inhale their concentrated exhalations from under the bed-clothes, run no small danger, and the danger is always increased or diminished in proportion to the supply of fresh air.

The following statement of Hancock forcibly illustrates the influence of fresh air on the prevalence of typhus: 'In the year '1819, I had occasion to see a very intelligent physician, con-'nected with one or two fever-hospitals in Dublin during the 'epidemic, who assured me that he had seen no proof of the 'existence of contagion in typhus, as it appeared in those institu-'tions under his care, where very great attention was paid to 'ventilation, and where the patients were not inconveniently 'crowded. But soon after this, I saw another physician, no less 'intelligent, who informed me that in the course of about four 'months, between two and three hundred persons were admitted 'into the Belfast Fever Hospital, and they were frequently so 'crowded in the wards as nearly to cover the floor with their beds, 'in which case, although the building is new, airy, and well 'regulated, the matron, twenty-two nurses, and the apothecary 'took the disease.'[o]

3. *Communication of Typhus by Fomites.* Notwithstanding Haygarth's statement to the contrary, it is highly probable that typhus may be communicated by fomites, or by apartments, or by articles of clothing strongly impregnated with the poison. Provided fresh air be excluded, such articles will retain the poison much longer than might be supposed.

There is good evidence in the writings of Pringle, Lind, Bateman, Jacquot, etc., that the typhus-poison can adhere to the walls of dwellings, to beams of wood, and to articles of furniture. Pringle gave an account of 23 persons, who were employed in re-fitting old tents in which typhus patients had lain, 17 of whom died of the infection.[p] Lind mentioned several instances, in which infected ships continued to impart the disease, long after the original sick had been removed;[q] and similar cases are recorded by Jacquot respecting the Crimean typhus. Many of

[o] Hancock, 1821, p. 339. [p] See Lind, 1763, p. 40. [q] Lind, 1763.

the transport ships brought infected troops from the Crimea, and disembarked them at Constantinople. A fresh set of passengers then embarked, who had not been exposed to the contagion of typhus; yet the disease appeared among them during the homeward voyage to France, without any reason to suspect that it had a spontaneous origin.[r] Bateman observed that successive occupants of the same dwellings in London often took typhus.[s]

The records of our prisons render it probable, that men who for months have not changed their clothes, and who have been living in close, ill-ventilated apartments, and on short allowances, may at length have their garments so impregnated with the poison of typhus, as to communicate the disease to others, without being themselves the subjects of it. John Howard found the English prisons in such a state, that his clothes became impregnated with the foul smell and retained it for hours after visiting them.[t] One of the most remarkable examples of typhus communicated by the clothes was the 'Black Assize' of the Old Bailey in 1750. Here the prisoners had not the disease which they communicated with such fatal effect to the court that tried them.[u] Lind mentions several cases, where a single person, though not ill himself, imparted fever by his clothes to a whole ship's crew. Foderé mentions a remarkable instance, in which the soldiers of the French army, during their retreat from Italy in 1799, communicated fever to the inhabitants of fifteen towns and villages where they halted on their route. The soldiers suffered from privations of every kind; they were ill-fed, their clothes were in tatters, their bodies were covered with filth and exhaled a noxious smell, and their shirts, unchanged for several months, were glued to the skin. Yet this same army was not attacked by fever, until it arrived at its destination and was massed within walls and under roofs. Soldiers also travelling *singly* did not communicate the disease.[x] Then, in our own day, there has been the notorious case of the Egyptian vessel, the Scheah Gehaad at Liverpool, the crew of which disseminated the poison of typhus by their clothes and persons, although they had not the disease themselves.[y]

Both Lind[z] and Trotter[a] state, that the nurses and porters at Haslar Hospital were well aware of the danger of contagion from piles

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[r] Jacquot, 1858, p. 99, 115. [s] Bateman, 1818. [t] Howard, 1784.
[u] Pringle, 1752; Heysham, 1782; Bancroft, 1811, p. 664.
[x] Foderé, *Méd. Légale*, tom. v. [y] Duncan, 1862. [z] Lind, 1763.
[a] Trotter, 1803, i. 177.

of infected clothes, and from cleaning the bedding of the sick, and that they were in the habit of measuring the amount of danger by the badness of the smell. The following case is recorded by Barker and Cheyne. 'A child, on being discharged from a Fever 'Hospital, was admitted into a charitable institution, and brought 'with her a small bundle of clothes, which had not been disinfected. 'The bundle was opened by a woman resident in the institution, 'who perceived an exceedingly disagreeable odour to issue from it. 'In a few minutes the woman became ill, and her stomach sick-'ened, which proved to be the beginning of a fever, such as was 'prevalent. Her's was the first case of the epidemic in the insti-'tution.'[b] Lastly, it has been a matter of common observation, that laundry-women, employed in washing the clothes and bedding of typhus patients, are liable to contract the disease, without having any direct communication with the sick.[c]

Woollen substances, as being the most prone to absorb and retain animal exhalations, are most fitted to transmit the typhus-poison. Haller of Vienna observes, that *dark*-coloured materials of clothing are more prone to absorb the contagion of typhus, and to convey it to other individuals, than those which are *light*-coloured. He found that among troops wearing dark-coloured uniforms, it more frequently happened, that new cases of typhus entered the hospital after a convalescent patient had rejoined his corps, than those wearing light or white uniforms. It may be mentioned, that Stork found that in dissecting rooms dark clothes acquired the cadaveric odour sooner, and were deprived of it less readily than light ones; and he ascertained, by experiments, that the absorption of odours is regulated by the laws which govern the absorption of light.[d]

Facts, like the foregoing, prove how highly reprehensible is the practice of employing street cabs for the conveyance of typhus patients.

Still it is satisfactory to reflect, that the poison must be highly concentrated to be transmitted by fomites, and that it is rendered inert by free ventilation. There are no instances on record where a medical man has been the medium of transmission of typhus to his patient or to his family, as sometimes happens in the case of scarlet fever. 'I have visited,' wrote Dr. Gregory, 'more than a 'thousand patients in fever—many of them, ten, twenty, or thirty 'times—yet I am certain I never brought the contagion into my

[b] Barker and Cheyne, 1821, i. 472 and ii. 139.
[c] Tweedie, 1833, p. 400; Henderson, 1843. p. 216. [d] Haller, 1853.

'own family'.[e] I am assured by Dr. Tweedie that on no occasion during his connection of thirty-three years with the London Fever Hospital, was he the medium of communication of fever.

4. *Length of exposure to the poison necessary to impart the disease.* There are many instances on record,[f] and several have come under my own notice, showing that if the poison be concentrated, the length of exposure sufficient to contract the disease may be very brief—in fact not more than a few minutes. If the poison be more dilute, it is probable that the chances of its taking effect are increased by protraction of the period of exposure.

5. *The latent period of the typhus poison* has been variously fixed as follows :—

Haygarth (1801) made the latent period			5 days to 2 months.
Hildenbrand (1811)	,,	,,	3 to 7 days.
Bancroft (1811)	,,	,,	1 day to 5 or 6 months.
Sir W. Burnett[g]	,,	,,	7 to 18 days.
Barker and Cheyne (1821)		,,	a few minutes to 6 wks.
Sir Henry Marsh (1827)	,,	,,	a few hours to as many weeks or months.
Dr. Gregory (1832)	,,	,,	10 days.
Dr. Perry (1837)	,,	,,	never less than 8 days.
Dr. Alison (1844)	,,	,,	very various.
Dr. Copland	,,	,,	3 to 14 days.
Huss	,,	,,	1 to 10 days.
Dr. Peacock (1856)	,,	,,	10 to 14 days.
Jacquot (1858)	,,	,,	9 to 11 days.
Barrallier (1861)	,,	,,	12 to 15 days.

According to my experience, the latent period is usually about nine days, but may vary from a few hours to twelve days. In Jacquot's cases, the latent period was calculated from the date of embarkation of healthy troops on board infected vessels, and was found to be nine to eleven days. In one of my attacks, I can fix the period of incubation with certainty at five days, as I saw no typhus patients after the one day of exposure, nor for many months before. There are many authentic instances where the latent period has been extremely short. The late Sir Henry Marsh[h] collected nineteen cases where the disease manifested itself almost instantaneously after exposure to the poison, and stated that he was acquainted with many others of a similar nature. In most of the cases, the persons complained of an offensive odour pro-

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[e] See CLARK, 1802.
[f] HAYGARTH, 1801, p. 65 ; MARSH, 1827.
[g] See GREGORY, 1832, p. 745.
[h] MARSH, 1827.

ceeding from the beds or bodies of the sick, and immediately suffered from head-ache, great prostration, nausea, or rigors, followed by the usual symptoms of typhus. Similar cases were mentioned by Haygarth;[i] others were observed by Gerhard, at Philadelphia, in 1836;[k] and two or three have come under my notice. In some of these cases, it might be difficult to exclude the possibility of previous exposure to the poison; but in others, there were no grounds for such suspicion; and, in all, the patients appeared to be conscious of the moment at which the poison entered the system. The poison of typhus, then, may be so concentrated, or the system so susceptible of its action, that its effect may be almost instantaneous.

On the other hand, cases of extreme protraction of the period of incubation must be viewed with some suspicion. I know no reliable case where it has extended beyond three weeks. Few, at all events, will agree with Bancroft in the opinion that an interval of five or six months may elapse between exposure to the poison and the commencement of the disease, an opinion to which he was forced by his determined opposition to the possibility of a spontaneous origin of the fever. There is one source of fallacy in the observations of some writers, who have extended the period of incubation to many weeks. In a body of men, who have been exposed to typhus, the disease may commence in some at the end of ten days, but in others, not until fifty or sixty days later. In the latter cases, the latent period cannot be legitimately calculated at two months because the persons first attacked become fresh *foci* for the propagation of the poison.

6. *Stage at which Typhus is most infectious.*—Haygarth mentions the case of a man who was said to have communicated the disease to his family before the fifth day; but he adds, that his information was less complete than he could have wished.[l] Hildenbrand was of opinion that the contagious poison was chiefly developed at the time of the appearance of the eruption, and that, as the eruption became petechial, the disease almost ceased to be contagious.[m] Dr. Perry of Glasgow was the first to advance the opinion that the period of convalescence is the most infectious in typhus. His statements are as follow: 'From numerous observations and experi-'ments, I am satisfied, that it (typhus) *is not contagious before the* '*ninth day*, perhaps not till a later period of the disease. Among 'many circumstances which establish this opinion, I may mention

[i] Haygarth, 1801, p. 65.
[k] Gerhard, 1837, xix. 299.
[l] Haygarth, 1801, p. 62.
[m] Hildenbrand, 1811, pp. 55 and 117.

'one experiment which I made upon a pretty extensive scale. The 'fever wards of the Glasgow Royal Infirmary are each capable of 'containing twenty patients. The beds are arranged in two opposite 'rows, and are pretty near each other. While the patients are in 'the acute wards they are not allowed the use of their clothes, 'though they may be able to sit up; they are, therefore, almost 'constantly confined to bed, excepting when rising to stool. Into 'the fever house are admitted cases of measles, scarlet fever and 'small pox; and patients are very frequently sent in, labouring 'under bronchitis, pneumonia, erysipelas, and other local inflam- 'matory affections. I found by experience, that when the latter 'class of patients were sent to the convalescent ward, where they 'necessarily mixed with the others, almost all those who had not 'had a previous attack of typhus fever were either seized with it 'before leaving the house, or returned soon after their dismissal, 'labouring under it; the period intervening between the time of 'their being sent to the convalescent ward and the attack, never 'being less than eight days. In consequence of these observations, 'I adopted the practice of not sending, as formerly, to the 'convalescent wards, those patients affected with inflammatory 'diseases, unless I ascertained that they were secured against 'typhus by having had a previous attack; but kept them in the 'acute fever wards till they were so far recovered as to go to their 'own houses; and the result was, after several months, that not one 'of those detained in the acute wards caught the disease while 'there, or returned with it afterwards.'[n] My observations at the London Fever Hospital confirm Dr. Perry's. I have often known typhus contracted by patients in the convalescent wards, but rarely, in the acute wards. The circumstance, however, has been probably due, not to typhus being most contagious during convalescence, but to the patients in the convalescent wards wearing their own clothes, which, before admission, were saturated with the typhus poison, and to their being brought into closer contact with one another. I am inclined to think that the disease is really most contagious, from the end of the first week up to convalescence, when the peculiar odour from the skin is strongest.

Whether typhus can be communicated by the dead body is a question of some importance, but not very easy of solution. Morgagni believed that there was some hazard in dissecting persons who died from fever, and mentioned a case of a prosector who died of a petechial fever contracted by dissecting the body of a female

[n] PERRY, 1836. No. 2. pp. 386-7-8.

patient.[o] Additional instances are mentioned by Rochoux.[p] But the difficulty in such cases is to exclude the chances of simultaneous infection from the living body. The following evidence from Dr. Roupell's[q] work on typhus deserves to be mentioned. At St. Bartholomew's Hospital, in 1838-9, the bodies of 17 persons who had died of typhus were dissected. Eight students were engaged upon each body, and many others were lookers-on. Six of the whole students at the Hospital took fever; but four of them had not dissected at all, and the remaining two were also exposed to contagion in the wards of the hospital. I may state, however, that, at the time of my first attack in Edinburgh, I had never entered the medical wards of the infirmary, nor seen a case of typhus, but that I dissected several hours a day in a close room, in which were many bodies of persons dead from typhus.

7. *Proportion of persons liable to be attacked by typhus.* If the poison be strong, the chances of escape are small, except in some cases of idiosyncrasy already alluded to (p. 67). Haygarth found that of 168 persons exposed to contagion, only 5, or 1 in 33, remained uninfected.[r] Whole families of eight or ten, comprising individuals of every age, are often attacked at one time. During epidemics, it has often been noticed, that all the nurses and hospital attendants on the sick are attacked, who have not had the disease before.[s] Of 22 hospital attendants in the service of M. Jacquot, every one took typhus.[t]

8. *Immunity from second attacks.* It is generally believed that typhus, like the exanthemata, attacks an individual only once in the course of his life. This opinion was expressed by Dr. Trotter, as the result of extensive experience of the disease among sailors.[u] It was likewise insisted on by Hildenbrand.[x] In 1837, Dr. Perry stated that typhus is taken only once in a lifetime, that a second attack is as rare as a second attack of smallpox, and rarer than a second attack of measles or scarlet fever. This conclusion was drawn from the circumstance, that since 1831, he had never known a patient re-admitted into the hospital with a second attack.[y] A similar opinion was expressed in 1840 by Stewart[z]; in 1843, by Henderson,[a] Cormack,[b] Wardell,[c] and others. In 1849, Dr. Jenner stated that he had never known the

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[o] Cook's *Morgagni*, ii. 592. [p] Rochoux, 1840, p. 157. [q] Roupell, 1839, p. 56.
[r] Haygarth, 1801, p. 32. [s] See page 80; also Tweedie, 1833, p. 400.
[t] Jacquot, 1858, p. 104. [u] Trotter, 1803, p. 213.
[x] Hildenbrand, 1811, p. 145. [y] Perry, 1836, No. 2, p. 386.
[z] Stewart, 1840, p. 300. [a] Henderson, 1843.
[b] Cormack, 1843. [c] Wardell, 1846, xxxix. 273.

same individual affected twice with typhus.[d] Drs. W. T. Gairdner[e] and Lyons,[f] both testify to the extreme rarity with which the same individual is attacked by typhus a second time. Jacquot took the precaution of employing hospital attendants in the Crimea, who had already had typhus, and in no instance found any have a second attack.[g] Indeed, the strongest argument in favour of acquired immunity, is derived from the fact, that nurses in fever hospitals, constantly exposed for a series of years to the poison of typhus, are never known to take the disease twice. I have been unable to discover any instance of a nurse at the London Fever Hospital having had typhus twice, although some have been there for many years, whereas fresh nurses during an epidemic, who have not had the disease before, are almost certainly attacked.

It is true, that many writers have mentioned instances of persons having two attacks of fever,[h] and cases are spoken of—those of two distinguished physicians in particular—where five or six attacks have occurred in the same individual. But after careful inquiry into the circumstances of many such cases, including the two specially referred to, I have obtained no evidence that more than one attack was true typhus. Even Irish physicians, who particularly refer to repeated attacks of fever in the same person, but who, for the most part, deny the plurality of continued fevers, admit that fever with a petechial eruption, rarely, if ever, attacks an individual twice.[i]

Still, there are undoubted examples of persons having more than one attack of typhus. In my own case (see p. 82), the characteristic eruption was well-marked, and the symptoms severe, on both occasions. The case of an Irish physician is also recorded, who had typhus twice, with the characteristic eruption in both attacks.[k] Dr. W. T. Gairdner mentions the case of a man who had a second attack of eruptive typhus after an interval of some years.[l] Jacquot admits that second attacks occurred in rare cases among the French soldiers in the Crimea, although he never met with an instance himself.[m] Lastly, M. Barrallier tells us, that of 698 prisoners who had typhus in the hulks at Toulon in 1855, nine took the disease a second time during the epidemic of the following year.

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[d] Jenner, 1849, No. 1, p. 38. [e] Gairdner, 1862, No. 2, p. 121. [f] Lyons, 1861, p. 213. [g] Jacquot, 1858, p. 225.

[h] See particularly Stokes and Cusack, 1848, iv., 138, v. 127; Douglas, 1845, p. 10; Stratton, 1847, p. 99; and Bartlett, 1856, p. 240.

[i] Barker and Cheyne, 1821, i. 241, and Bartlett, 1856, p. 240.

[k] *Irish Report*, 1848, vii. 399. [l] W. T. Gairdner, 1859, p. 242

[m] Jacquot, 1858, p. 224.

It is not stated, that the eruption was present in both attacks; but in seven, the first attack was slight, the second severe; and in one, both attacks were severe.[n]

But such cases are probably not more common than second attacks of scarlatina or variola. Even of small-pox, I know the particulars of a case, where death resulted from a third attack.

9. *Specific Gravity of the Typhus-Poison.* According to Haller of Vienna, the contagious principle of typhus is lighter than atmospheric air. Ozone, when admitted into a fever ward, was ascertained to become first lost in the upper regions of the atmosphere. Moreover, when the under stories of a hospital were filled with typhus patients, those in the upper stories were always observed to become infected, when there was a communication between the air of the two stories. On the contrary, when only the upper stories contained cases of typhus, the patients in the under part of the house enjoyed perfect immunity.[o]

10. *Effect of Heat on the Typhus-Poison.* Henry, proved by experiment, the destructive influence of heat over the specific poisons of several of the exanthemata. The vaccine virus failed to take effect after exposure for some hours to a dry heat of 130° Fahr. In four different instances, flannel waistcoats, taken from patients labouring under scarlet fever, were exposed for some hours to a dry heat of 204° Fahr., and were then worn with impunity by children who never had the disease. Three flannel jackets were taken from a typhus patient and exposed for some hours to a temperature of 204° Fahr. One was kept under the nostrils of a person in health for an hour and three quarters; a second was worn next the body of the same individual for two hours; while the third was shut up in an air-tight canister for some days, and then kept for some hours within twelve inches of the face of the same person, a current of air being directed across the flannel to the face. No result followed, although the person had been fasting for eight hours and was much exhausted by disease, so as to predispose him to typhus.[p] These observations, although insufficient for the purpose of scientific demonstration, afford strong presumptive evidence that dry heat is a powerful disinfectant agent. Owing, no doubt, to their doubtful propriety, Dr. Henry's experiments have not been repeated; but where the principle advocated by him has been acted on, the results have been satisfactory.[q]

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[n] Barrallier, 1861, p. 370. [o] Haller, 1853, p. 262. [p] Henry, 1831.

[q] See *Report of aComm. of York. Med. Soc.* to investigate the disinfectant power of heat. *Brit. Med. Journ.*, Ap. 7. 1860, p. 272.

2. *Spontaneous Origin.*

Although in a large proportion of cases of typhus, especially during epidemics, the specific poison is derived from persons previously infected (contagion), it is, I believe, equally true, that in other cases it is generated spontaneously. This is a statement which many will be prepared to contest; but, in my opinion, the facts in favour of it admit of no other explanation. The conditions under which the poison is developed *de novo* are overcrowding of squalid human beings with deficient ventilation: in other words, the poison is generated by the concentration of the exhalations from living beings, whose bodies and clothing are in a state of great filth.

The intimate connection between the prevalence of typhus and overcrowding has been already demonstrated, and is generally admitted. But the fact of typhus being confined to overcrowded localities admits of explanation, on the supposition that it always originates by contagion. It is obvious that, during an epidemic, all possible sources of contagion can rarely be excluded from houses situated in the centre of a large town. Still, it is worth observing, that typhus patients have often been admitted into the London Fever Hospital, who stated that there had been no previous cases of illness at their homes, who denied having been exposed to any contagion,[r] and who could attribute their disease to no other cause than to having been one of eight, ten, or even seventeen adults, who had slept for many weeks in one small room of a house situated in a narrow court.

But more conclusive proofs of typhus being generated spontaneously is derived from a study of the mode of origin of sporadic cases in the absence of any great epidemic, and of outbreaks in public institutions and in isolated bodies of men.

a. Mode of origin of sporadic cases and of limited outbreaks of typhus.

In 1781, Dr. J. Heysham traced the origin of an outbreak of typhus at Carlisle to a house inhabited by half a dozen poor families. In order to reduce the window tax, every window that even poverty could dispense with was built up; and all sources of ventilation were thus removed. The smell in this house was overpowering and offensive to an unbearable extent. There was no evidence that the fever was imported into this house; but thence it was propagated to other parts of the town (see page 82), and fifty-two of the inhabitants died of it.[s]

[r] See also *Irish Report*, 1848, viii. 305.

[s] Heysham, 1782.

About the same time, Dr. John Hunter, physician to the army, recorded an instance of typhus in a family in London. The family consisted of father, mother, and several children; they were very destitute and were lodged in a room, not exceeding twelve by fourteen feet square. Typhus was not prevalent at the time, and in this instance it could not be traced to contagion.[t]

In 1836, an epidemic of typhus appeared at Philadelphia, where it had been unknown for years. The disease originated in a very crowded part of the town. 'Amongst the very first cases were seven negroes, the entire population of a cellar.'[u]

In 1843, an epidemic of typhus occurred at Broulhac, an elevated spot in the Canton de Puy in France. It differed from the ordinary fever of the country in being very contagious. The symptoms were those of typhus, viz.:—Dull, heavy expression, constipation, dry brown tongue, subsultus and delirium; petechiæ and occasionally parotid buboes; after death the intestines were found to be sound. Of the 118 inhabitants, 45 were attacked and 9 died. Starvation and overcrowding were the alleged causes. The first cases were traced to a house, where there was overcrowding and no ventilation. One part of the village where the houses were of a better sort, remained exempt. There appeared no possibility of imported contagion, for the report of the epidemics of France, from which this account is extracted, made no mention of the prevalence of typhus elsewhere.[x]

In 1859, typhus, after having disappeared from Edinburgh for some months, (see page 52) again became prevalent, 30 cases being admitted into the Royal Infirmary from May to August. The localities whence they were derived were investigated by Dr. W. T. Gairdner; they were in the worst and poorest parts of the town, and in regard to several it is stated, that the disease appeared under circumstances of extreme over-crowding and deficient ventilation.[y] There was no evidence that the disease was imported into Edinburgh at this time.

The following cases were carefully investigated by myself.

From April 20th, 1858, to March 12th, 1859, inclusive, only two cases of typhus with the characteristic eruption were admitted into the London Fever Hospital, although in 1856, the number, had amounted to 1062. In March, 1859, seven well-marked cases were admitted from one house, 10, Meridian-place, Ber-

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[t] HUNTER, 1785. [u] GERHARD, 1837, xix. pp. 294–7.
[x] *Mém. de l'Acad. de Méd.* tom. xiv. p. 47. [y] W. T. GAIRDNER, 1859, p. 243.

mondsey. It became an interesting point, to investigate the precise conditions under which this fever appeared. The following account is drawn up, partly from enquiries made by myself on the spot, and partly from a communication, for which I am indebted to Dr. Challice, the Medical Officer of Health for the district.

1. The court, in which the house was situated, was paved and open at both ends, and was about eleven feet wide. The drainage in the court was satisfactory. In fact, only a year before, great improvements had been carried out. All the cesspools had been emptied and filled up; the drains trapped, and the water let on. The privy in No. 10, was furnished with a soil-pan and trapped, as was also the sink. These facts are important, inasmuch as the fever was not that which results from the putrid emanations from drains.

2. The house, No. 10, consisted of two floors, connected by a very narrow staircase. There were two rooms on each floor: and in each room, a door, one window, and a fire-place. All the rooms were little better than closets, their dimensions being as follows:

	Length.	Width.	Height.	No. of Cubic Feet.[z]
	Ft. In.	Ft. In.	Ft. In.	
1. Ground Floor—Front Room	8 9	8 6	8 0	595
Back „	8 6	8 0	8 0	544
2. Upper Floor—Front Room	11 2	8 6	7 2	680
Back „	8 6	8 2	7 2	497

The doors of the rooms on the ground floor opened into a passage not more than two feet wide. The windows in all the rooms could be opened; but throughout the winter, and up to the outbreak of fever, they had been always shut.

3. A mother with her six children occupied the two rooms on the ground floor. The mother was aged 34; and the respective ages of the children were 18, 17, 15, 10, 7, and 3. Three slept in one bed, in the front room; and four, in the back room. After the fever broke out, the grandmother of the children came from Dover to nurse them, and she also slept in one of the rooms. The rooms upstairs were occupied by a man and his wife.

4. It will thus be seen, that before the arrival of the grandmother seven human beings occupied 1139 cubic feet of space, or each individual had only 163 cubic feet. After the arrival of the grandmother there were only 142 cubic feet to each.

5. There were no means of ventilation. Dr. Challice described the rooms as having the 'peculiar animal odour always noticed

[z] In this and the following instances, no allowance is made for the space occupied by the furniture.

'in cases of overcrowding.' The habits of the family were filthy in the extreme. The parish inspector found the rooms 'alive with vermin'; and the nurses in the Fever hospital declared, that they had scarcely ever known patients admitted in such a filthy condition.

6. The father of the family was a sailor, and had been at sea for many months; and although the family were not absolutely penniless, the mother spent most of their little earnings in gin.

7. There were no other cases of fever in the court or in the immediate neighbourhood. Indeed, true typhus was at the time extremely rare throughout the metropolis. None of the members of either family had, as far as could be ascertained, been exposed to any contagious disease. Shortly afterwards, however, several cases of typhus occurred in the next house, and two were admitted from it into the London Fever Hospital.

8. The mother and eldest child were first attacked about the end of February. Three of the other children were seized during the first week of March, and a fifth in the second week. The sixth child, the youngest, escaped. The mother and five children were admitted into the Fever Hospital on the 12th and 15th of March. All recovered. The grandmother, who came from Dover early in March, took the fever, and died on the 15th, at 10, Meridian-place. The man, who resided up-stairs, was taken ill (contagion?) about the 9th of March, was admitted on the 15th, and died on the 22nd. His wife did not take the fever.

The next cases of typhus admitted into the London Fever Hospital came from No. 5, Henry-passage, St. Pancras. The following is the sequence of events concerning them:—

1. The fever first appeared in a family residing on the ground floor, and consisting of a father, aged 54; a mother, aged 40; and six children, of the respective ages of 16, 14, 12, 10, 8, and 5.

2. These eight persons resided and slept in two rooms, which together contained only 1378 cubic feet of space, making an allowance of only 172·5 cubic feet to each individual. This I ascertained from personal examination. Each of the two rooms was furnished with a door, one window, and a fire-place. The mother informed me, that during the winter, and previous to the outbreak of fever, the windows had seldom been opened.

3. The whole family had long been very destitute, the father having for many months been out of employment.

4. No source of contagion could be traced. These were the first cases of typhus in the court and in the neighbourhood. But, on the other hand, they formed a focus of contagion, whence other

cases originated. Shortly after, cases appeared in the next house, one of which was admitted into the Fever Hospital. One of the mother's sisters, came from an adjoining street, to attend upon her. She caught the fever, as did also her husband and child; and all three died. A third sister came to nurse this last one, from another street in the neighbourhood. She was taken ill shortly after with fever, as were also her husband and child. The husband died. Here, indeed, was a melancholy instance of the results, which may accrue from a neglect of sanitary precautions in a single family.

Again, after a complete absence of typhus for six months, several cases occurred in the spring and summer of 1860.[z] I visited the localities whence all the first cases came.

Several came from a court at Limehouse, where the fever originated in an underground cellar, containing 912 cubic feet of space, with one window, which was never opened. This cellar was inhabited by eight persons (114 cubic feet to each), who were in a state of great destitution. There had been no fever before in the court or neighbourhood; but from this cellar it spread by contagion to several other houses in the same court.

Another group of cases came from Pump Court, White Horse Alley, Holborn. A family consisting of father, mother, and four children of the respective ages of 18, 15, 11, and 9, inhabited a room on the ground floor, whose dimensions were 10 feet 5 inches broad; 12 feet, 3 inches long; 8 feet, 3 inches high, making 1072 cubic feet. All six slept in this room, so that each had only 178 cubic feet of space, which was still further diminished by a great accumulation of furniture, consisting of two large beds raised two and a half feet from the floor, a chest of drawers, several tables and chairs, and a number of boxes. In the night, when the beds were let down, the floor was literally covered with furniture. There was one door and one window; the door was always shut at night and the window shutters closed. The window looked into a court, a yard and a quarter wide, on the other side of which was a high wall, and beyond this, a range of high houses. The family had resided in this

[z] It has already been stated, that sporadic cases of typhus often become more common at the end of spring, or at that period of the year in which the dwellings of the poor have been longest subjected to overcrowding and deficient ventilation. When the poison is once generated, it may continue to spread through the summer by contagion, but, by the end of summer, the effects of ventilation have had time to come into play, so that in autumn the disease may entirely disappear.

house for many months, and had latterly been in very reduced circumstances, owing to the father being out of work. Four of the six took typhus, which at the time was unknown in the neighbourhood, and indeed was only met with in one or two distant localities throughout the metropolis.

In a third case investigated, the circumstances were very similar.

Now, in the above cases, it may be argued, that we cannot be certain that the disease was not primarily introduced by contagion. But to this objection it may be replied, that at the periods in question there were no cases of typhus in the immediate neighbourhood; that no member of the families first affected had been exposed to contagion; and that typhus was scarcely to be met with, either in the metropolis, or in any part of England. If the spontaneous origin of typhus in these cases be objected to, it must be admitted, that the specific poison is always and everywhere present, ready to take effect, whenever (and only when) the causes, supposed to generate it, are present.

b. Jail Fever (See *Synonyms*, page 19).

The disease, which was formerly so prevalent in our prisons, and which was described as the 'Gaol Fever' and the 'Jayl Distemper,' was Typhus. Many observations show that it originated in the prisons; and it was the general belief that the cause was overcrowding, with deficient ventilation. The prisons, indeed, constituted the principal foci, whence the disease spread with dire results among the population. Such was the story of the various 'black assizes,' of which history furnishes us with an account of six. A brief notice of these assizes may not be out of place, although our knowledge of some of them is very meagre.

The first occurred at Cambridge, during the Lent Quarter Sessions in 1522, the thirteenth year of the reign of Henry VIII. The justices, gentlemen and bailiffs, and most of the persons present in court were seized with a fever which proved mortal to a considerable number. No account is preserved of the symptoms of this fever; but the circumstances were similar to those of subsequent black assizes, in which the disease was undoubtedly typhus.[a]

The year 1577, or twentieth of the reign of Queen Elizabeth, was notorious for the Oxford 'black assize.' This assize was held at Oxford Castle on July 4th and two following days, for the trial of Rowland Jencks, a bookbinder and a Roman Catholic, for treason and profanity of the Protestant religion. Jencks was not

[a] Ward, 1758, p. 703.

the only prisoner brought before the court; but the accounts state, that, after judgment was pronounced against him, 'an infectious damp or breath' arose among those present. Many seem to have been taken ill on the spot, including Sir Robert Bell, chief Baron of the Exchequer, Sir Nicholas Barham, Serjeant at Law, two sheriffs, one knight, five justices of the peace, and most of the jury, of whom several died within a few days. 'Above 600 'sickened in one night; and the day after, the infectious air 'being carried into the next village, sickened there an hundred 'more.' On the 15th, 16th, and 17th of July, 300 more fell sick; and between the 6th of July and the 12th of August, 510 persons perished. The following are mentioned as the symptoms: loss of appetite, great headache, sleeplessness, loss of memory, deafness and delirium, so that the patients would get up and walk about like madmen. The general impression at the time was, that the 'infection arose from the nasty and pestilential smell of the 'prisoners when they came out of the jail, two or three of whom 'had died a few days before the assize began,' the only other explanations offered being, that it resulted from the 'diabolical 'machinations of the papists,' or, according to the catholics, that it was a miraculous judgment on the cruelty of the judge, for sentencing the bookbinder to lose his ears.[b]

In 1586, another black assize occurred at Exeter. Some time before, thirty-eight Portuguese seamen had been cast into 'a deep 'pit and stinking dungeon' in Exeter Castle. They had no change of raiment, and were left to lie upon the bare ground. A contagious fever broke out among them, which, from Hollingshed's description, was evidently typhus. Many of them were sick during their trial, and by them the disease was communicated to those present in the court. The judge, three knights, and many others died, and the disease spread over the whole county. In this instance, very few became ill until fourteen days after the

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[b] See WARD, 1758, p. 699. BANCROFT, 1811, p. 653; also WOOD, *Hist. and Antiq. of the University of Oxford*, 1796, ii. 188; SIR R. BAKER'S *Chronicles of the Kings of England*. Lond. 1665, fol. p. 353; and STOW'S *Chronicles*, Lond. 1592, p. 681.

Bancroft maintained that the disease in this instance was not typhus, and laid much stress on the statements in some of the accounts, that it was not contagious, and that none but those present in the court were attacked. But these statements, if true, would not be opposed to what is known of the effects of dilution upon the typhus-poison. (See page 86). Bancroft also argued that the typhus-poison could not take effect so rapidly as in this instance, an argument which is now known to be without foundation. (See page 89).

trial. The fever was believed to have proceeded from 'contagion 'by reason of the close aire and filthie stinke of the gaole.[c]

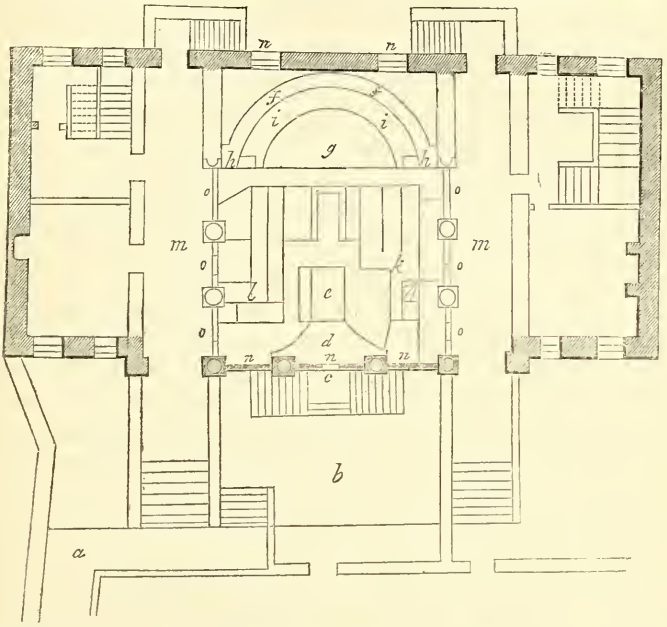
There are accounts of a fourth 'black assize' at Taunton, during Lent in 1730. A contagious fever was communicated by the prisoners, who had been removed from Ilchester jail, to the judges and many others present in court. The Lord Chief Baron, the Serjeant at Law, and the High Sheriffs of Somersetshire, all died of the disease, which spread widely at Taunton, and proved fatal to several hundreds.[d]

Twelve years later, there was a fifth black assize at Launceston, an account of which is contained in the writings of Huxham. 'A putrid contagious, and highly pestilential fever, which had 'been *generated in* the prisons,' was widely disseminated by means of the county assize, and occasioned great mortality. Among the symptoms were—great prostration and oppression, a florid rash with petechiæ, watchfulness, delirium, tremors, subsultus, black dry tongue, and fetid breath. The pulse was weak from the commencement, even in the robust, and 'bleeding killed the patient, 'and not the disease.'[e]

The sixth and last 'black assize' was that of the Old Bailey, in April 1750. Nearly a hundred prisoners were tried, who were all, during the sitting of the court, either placed at the bar, or confined in two small rooms, which opened into the court. The court was crowded to excess, and many present were 'sensibly 'affected with a very noisome smell.' Within a week or ten days, many of those present were seized with a 'malignant fever,' among the symptoms of which were a weak pulse, delirium and petechiæ. Its duration was a fortnight. That this was the jail distemper or typhus, appears from a pamphlet published at the time by Sir John Pringle. More than forty persons died of it, including the Lord Mayor, two of the judges, an alderman, an under-sheriff, and several of the jury. In less than six weeks the disease disappeared. It is uncertain whether it was communicated by the sick to any, who had not been present in the court. A remarkable circumstance is, that those who were situated *highest* in the court, as the Lord Mayor, Judges and Middlesex Jury, and those in the gallery on the left hand of the court, were chiefly infected with the fatal poison. This was attributed by Dr. Stephen Hales, F.R.S., to a wide sash-window, on the left hand side facing the judges being left open, through which an easterly

[c] Bancroft, 1811, p. 661. [d] *Gentleman's Magazine*, May 1750.
[e] Huxham, 1752, vol. ii. p. 82.

wind entered, 'blowing down the most venomous vapour which 'was near the ceiling,' against the persons chiefly attacked. It is also to be noted, that neither the prisoners under trial, nor any in the jail were suffering at the time from typhus.[f] A plan of the Old Bailey, copied from Bancroft's work, is here annexed.



a. Passage from prison to court. *b*. Bail-dock for prisoners before trial. *c*. Door under window into court. *d* and *e*. Prisoners' Box, etc. *f*. Bench for Lord Mayor, Judges, and Aldermen. *g*. Table for Counsel. *h*. Boxes for Sheriffs. *i*. Bench for Counsel. *k*. Middlesex Jury. *l*. London Jury. *o*. Doors. *n*. Windows. The one facing the judges, on the right hand of page, was open during the trial.

Such events are not surprising, when one studies the frightful pictures drawn by John Howard of the state of our prisons in former days.[g] 'My reader,' said Howard, 'will judge of the 'malignity of the air of gaols, when I assure him that my clothes 'were, in my first journeys, so offensive, that, in a post-chaise, I 'could not bear the windows drawn up, and was therefore often 'obliged to travel on horseback. The leaves of my memorandum 'book were often so tainted, that I could not use it until after 'spreading it an hour or two before the fire.' Howard likewise recorded many instances, where the fever appeared to be generated by over-crowding, with a want of fresh air and cleanliness. For example, he related how seventeen women, being confined in a room in the Cambridge Bridewell, without any fire-place, the air

[f] See FOSTER, 1762, p. 74; PRINGLE, 1750, 1752; HEYSHAM, 1782; BANCROFT, 1811, pp. 140, 664.

[g] HOWARD, 1784, and 1789.

soon became 'extremely offensive and occasioned a fever among 'them' which proved fatal to three or four. The opponents[h] of the spontaneous origin of typhus, put much stress on the following statement of Howard: 'If it were asked what is the cause of the 'gaol fever, it would in general be said, the want of fresh air and 'cleanliness. But as I have seen, in some prisons abroad, cells 'and dungeons as offensive and dirty as any I have observed in 'this country, where, however, the distemper was unknown, I am 'obliged to look out for some additional cause for its production.' But Howard did not seem to doubt that the fever originated in the prisons, nor did he hint that the poison was imported. All that he said respecting the additional cause is expressed as follows: 'I am of opinion that the sudden change of diet and 'lodging so affects the spirits of new convicts, that the general 'causes of putrid fevers exert an immediate effect upon them.' (p. 231). Moreover, Howard did not say that the prisons, visited by him on the continent, were at that time over-crowded, but merely 'offensive and dirty,' conditions which are not believed to generate typhus; while his statement, that the jail fever is peculiar to Britain, is now known to be an error. The public opinion resulting from Howard's investigations was thus expressed, in the preamble to an Act of Parliament, passed soon afterwards: 'Whereas the malignant, commonly called the Jail Fever, is 'owing to a want of cleanliness and fresh air, be it enacted, etc.'[i] Thanks to the philanthropic labours of Howard, the sanitary condition of English prisons is now so perfect, that typhus can seldom be said to be generated in them.

But so late as 1815, Harty showed that typhus was constantly generated in the prisons of Dublin. It always appeared after over-crowding. The convicts in the Dublin Newgate were allowed to accumulate for twelve or twenty months, and were then transported. Typhus always broke out among them shortly before each embarkation, and only then. It was not due to importation, for the convicts had little or no communication with the public, and the disease did not appear at the periods in question in another class of prisoners in the same building, who had free communication with the public, but who were not over-crowded.[k]

Again, during the present century, many epidemics of typhus have occurred in jails on the continent of Europe, under circumstances the same as those in which the disease appeared in our own prisons, before the time of Howard. In the early part of the

[h] Bancroft, 1811, p. 149; Watson, 1843, vol. ii.
[i] Alderson, 1788, p. 7. [k] Harty, 1820, pp. 161 and 282.

century, these outbreaks were very common. The epidemics in the prisons of Nantes and Auxerre, were attributed to over-crowding and deficient food, while that at Posen commenced in the prison, and spread over the town.[l]

In 1839-40, an outbreak of fever occurred in the jail at Rheims, which resembled typhus in most of the symptoms, and which differed from the ordinary fever of France in being eminently contagious. Of the attendants on the sick, thirty-five were attacked. All the Sisters of Mercy who had typhus in 1814 escaped, but several who had passed through enteric fever, had now most severe attacks. There was no fever of a similar kind at Rheims, nor probably indeed in France. According to Landouzy: 'L'encombrement des prisons doit donc être regardé comme la 'cause déterminante de l'epidémie de Reims.' The number which one part of the jail was calculated to hold was eighty, or at most a hundred, and although it had been the custom to admit so many as 120 or 140 prisoners, the number had been raised to 190, a month or two previous to the outbreak of the fever. The cells in which the prisoners were confined during ten out of the twenty-four hours, were only large enough for three persons, but were made to contain sixteen. Moreover, the fever commenced in, and was confined to, the overcrowded cells: only two cases occurred in the building allotted to condemned prisoners, who were not over-crowded; while the female department escaped entirely.[m]

Lastly, in 1854, an outbreak of typhus occurred in the jail at Strasbourg. From 1814 to 1840, the prison had been remarkably healthy; but from that date, owing to a change in the diet, scurvy began to prevail, but still there was no typhus. 'La maladie,' says 'Forget, s'est developpée sous l'influence de l'encombrement, le 'chiffre des détenus ayant été porté de 340 à 360 en moyenne à 780.' That the disease was true typhus, was proved by the entire clinical history, and by the absence of any intestinal lesion after death. Before this, typhus may be said to have been unknown at Strasbourg, since the wars of the first Napoleon. In 1841, when Forget wrote his work on the ordinary fever of France, he does not appear to have seen a case of typhus; but in 1854, he at once recognised it as a new disease, and hastened to communicate to the French Academy proofs of the non-identity of typhus and typhoid fever.[n]

From these, and many other instances, it follows, that whether

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[l] Gaultier De Claubry, 1838, *ed.* 1844, pp. 48, 61, and 81.

[m] Landouzy, 1842. The symptoms and *post-mortem* appearances of the fever at Rheims will be referred to subsequently. [n] Forget, 1854.

in England or on the Continent, the circumstances under which the jail fever appears are always the same, while every conceivable source of importation is often excluded.

c. Ship Fever.

During last century, typhus was a very common disease on board ship, and was known as the 'Ship Fever,' and the 'Infectious Ship Fever.' Dr. James Lind, Physician to the Fleet, although he believed that the disease was often traceable to contagion, added that it was for the most part confined to the small vessels of the fleet, and mentioned several instances wherein he considered the fever to have originated *de novo* from overcrowding on board ship. One was that of the 'Diana' frigate, in which typhus appeared at sea, several weeks after leaving the coast of America. 'Thus,' he said, 'a seasoned crew became in-'fected, as it would appear, from the closeness or damp below, 'occasioned by the hatchways being kept shut in consequence of 'a storm.'[o] Many similar observations were made by Dr. Thomas Trotter,[p] and by Sir Gilbert Blane, who served in the British Navy under Admiral Rodney, and who thus summed up the results of his experience: 'The infection of fever is not always imported from 'without, but may be originally and spontaneously generated on 'board. The causes of this are want of personal cleanliness, and 'also confinement and crowding in close apartments.'[q] Nor were these observations confined to British vessels. M. Fonssagrives, in his account of the importation of typhus into the town of Brest in 1758, observes: 'Rien n'etait d'ailleurs plus habituel, dans 'ces temps calamiteux, que de voir l'encombrement, la misère, les 'privations, le sacrifice de tous les intérêts de l'hygiène aux exi-'gences irresistibles de la guerre, engendrer le typhus, au sein des 'équipages. La plupart des épidémies de fièvre grave, dont les 'annales de la navigation ait conservé le sinistre souvenir, n'ont 'été autre chose que des irruptions du typhus, à bord des navires 'mal tenus, humides, et encombrés.'[r] The following are a few more modern examples of the appearance of typhus on board ship, independently of importation.

In the spring of 1810, typhus broke out among the French prisoners confined in the prison-ships in Plymouth harbour. Typhus was not prevalent in Plymouth; and, even if it had been, the seclusion of the prisoners could not have been more complete. But on board, in addition to a spare diet, and the mental depres-

[o] Lind, 1763, p. 25. [p] Trotter, 1803, i. 181; and iii. pp. 151, 153, etc.
[q] Blane, 1789, third ed. 1803, p. 228. [r] Fonssagrives, 1859, p. 243.

sion consequent on their situation, the prisoners were packed together in a most shameful manner. For thirteen hours out of the twenty-four, upwards of 400 of them were crowded into a space measuring 60 feet by 42 feet, and only $4\frac{1}{2}$ feet high. The only ventilation was through the port-holes, which were almost closed by thick iron gratings; and the air was so dense, that a lighted candle appeared in it as through a thick mist. Such was the condition of the prisoners for some time before the commencement of the epidemic. Of 4,000 persons, 1,050 took typhus, and 150 died of it.[s]

In the winter of 1829-30, an epidemic of typhus broke out on board the French convict hulks at Toulon. The disease was unknown in Toulon, there not being a single case, even among the workmen in the harbour. That it was really typhus, and not the ordinary *Fièvre typhoide* of France, was proved by the symptoms and *post-mortem* appearances. 'Jamais on n'a rencontré l'exan-'thème intestinal qui appartient à la dothinenterie.' The origin of the epidemic was attributed to over-crowding and deficient food ('l' encombrement d' hommes mal nourris').[t]

Five other epidemics of true typhus have been observed in these same hulks at Toulon—in 1820, 1833, 1845, 1855, and 1856. The disease has quite disappeared in the intervals, has never prevailed in the town of Toulon, and for the last forty years has been scarcely known throughout France. M. Barrallier, the present professor of Pathology in the Naval School of Toulon, thus writes respecting them: 'L'encombrement a toujours été considéré comme 'la cause principale et déterminante de la maladie.' Among the accessory causes were deficient food, over-fatigue, and want of personal cleanliness.[u]

Several instances are mentioned by Jacquot, where typhus seemed to originate from over-crowding on board the French ships employed in transporting troops from the Crimea. With regard to some, the introduction of the poison by fomites is barely possible; but concerning one, M. Godélier averred to the French Academy: 'Ce typhus est né sur le Monarque, et du 'Monarque même.'[x]

During the late war in Italy, typhus made its appearance in a French vessel, 'L'Entreprenante,' carrying troops from Algeria to the Adriatic. The men were all in perfect health on leaving Algeria, where typhus is probably unknown. 'Tous ont 'rapporté à l'encombrement seul la cause de la maladie.'[y]

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[s] De Claubry, 1838, ed. 1844, p. 37. [t] Fleury, 1833; Keraudren, 1833.
[u] Barrallier, 1861, p. 189; Anon. 1833, p. 480. [x] Jacquot, 1858, p. 76; Godélier, 1856, p. 885. [y] Barrallier, 1861, p. 35.

Lastly, there is the remarkable case of the Egyptian frigate, 'the Scheah Gehaad,' the crew of which imported typhus into Liverpool in 1861. Three persons took typhus who went on board the vessel in the docks. The crew likewise communicated typhus to three of the attendants at the public baths, and to twenty-five persons in the Southern Hospital. This crew consisted of 476 persons, mostly Arabs. During the lengthened voyage of thirty-two days from Malta, the weather was cold and stormy; and the men, unaccustomed to the rigour of a northern winter, and not provided with suitable clothing, crowded below for warmth and shelter. Even they, whose turn it was for duty, had to be driven up on deck. The space below deck was 'quite 'insufficient for so large a number,' and there was 'no attempt to 'promote ventilation.' The persons and clothing of the men were filthy in the extreme; and they discharged the contents of their stomach and bowels in all parts of the ship, which on arriving at Liverpool, was so offensive that it had to be sunk in the graving dock. Moreover, 'the rations served to the men 'were much below the proper standard, as regards quantity,' and the crew suffered from mental depression and over-fatigue, consequent on the boisterous weather. It is important to notice, that neither during the voyage, nor in Liverpool, had any of the crew the fever, which they communicated to others.[z] (See page 87).

d. Military Fever.

But again, typhus is a disease as old as the disputes of nations, and is a constant accompaniment of warfare. Its characters are recognized in the descriptions handed down to us of the majority of those epidemics, which have decimated the ranks of armies in the field, and the garrisons of besieged cities. From this circumstance, indeed, many of its appellations, such as Camp and Army Fever, Kriegspest, etc., are derived, (see *Synonyms* p. 19). The circumstances under which it has appeared have been invariably those of over-crowding, with bodily and mental depression; and it is especially to be noted, that in many parts of the continent of Europe, where typhus never occurs in time of peace, it becomes epidemic in time of war. Ample illustrations are found in the histories of the campaigns of Maximilian II., in Hungary; of Francis I., in Italy; of Charles V., and Charles XII.; of Louis XIV., of Frederick the Great, and of the first Napoleon; and in those of the late Crimean war.

One of the best works on typhus ever written, is that of J. V. Hildenbrand,[a] whose experience was mainly derived from an epi-

[z] DUNCAN, 1862. [a] HILDENBRAND, 1811, pp. 31, 306.

demic at Vienna, which followed the campaign of 1806. His observations led him to divide typhus into *communicated* and *original*. In the latter, he considered that the poison was generated *de novo* by air, 'trop chargé d'exhalaisons humaines,' and that, thus produced, it could afterwards spread by contagion.

But in order to realise to the fullest extent the baneful effects of over-crowding and deficient food, we must study the heart-rending accounts of the sieges of Saragossa[b] and Torgau,[c] and of the garrison of Dantzic, in 1813.[d] In 1813, there perished by typhus in Dantzic, two-thirds of the French garrison, and one-fourth of the population. 'La cause vraiement de ce typhus,' said M. Tort, 'fut donc évidemment la réunion d'un trop grand nombre 'd'hommes dans les lieux trop étroits.'

The fearful extent to which typhus ravaged the French and Russian armies in the Crimea, is fresh in the memory of all. The disease was not endemic in the Crimea, and no evidence has been adduced to show that it was imported; but its origin was universally attributed to over-crowding and deficient food. In the winter of 1854-5, the commissariat and lodgment of the English troops were very inferior to the French, and the English suffered most from typhus. But in the next epidemic of 1856, the tables were reversed. The English army, now provided with large and airy huts, were almost exempt from typhus; but of the French 12,000 were attacked, of whom 6,000 died. Here is Jacquot's description of the lodgments of the French at this time. 'Après un séjour 'prolongé dans la boue des tranchées, après les factions, les tra-'vaux, les corvées, les marches dans les champs profondément 'défoncés, après avoir été mouillés par la pluie et la neige, les 'soldats grelottants et manquant le plus souvent d'effets de 'rechange, s'entassent sous les tentes et les huttes, allument, s'ils 'peuvent, quelque maigre feu, et ferment hermétiquement toutes 'les ouvertures, avec une persévérance et une insistance contre 'lesquelles échouent les conseils les plus pressants et les mesures 'les plus sévères. L'extrême malpropreté des hommes, les haleines 'fétides, la fumée du tabac, l'évaporation de l'eau qui trempe les 'vêtements, tout se réunit pour empester ces bouges étroits. Là 'est le typhus: au dehors est la congélation poussée souvent jus-'qu'au sphacèle complet des pieds. Le danger se montre partout, 'mais le pire est au dedans. L'encombrement est général. *Dans les 'ambulances strictement calculées à* 200 *ou* 400 *hommes, on en accumulé 'le double et parfois le triple.*'[e] Here then, were two armies, in the

[b] Gaultier de Claubry, 1838, ed. 1844, p. 33. [c] Ibid. p. 43.
[d] Ibid. p. 41. [e] Jacquot, 1858, p. 65.

immediate vicinity of one another, with typhus prevailing first in the one, and then in the other, in a direct ratio to the extent of privation and over-crowding. The French surgeons could arrive at but one conclusion. Adolphe Armand stated: 'Dans cette épidémie, la 'cause première, l'encombrement, est une chose évidente.'[f] M. Scrive, officer of health, observed that typhus differed from other contagious diseases, inasmuch as it 'prend naissance à la suite de 'la modification profonde, qui s'opère dans l'organisme humain, 'sous l'action continue des fatigues excessives, la misère, l'alimenta-'tion insuffisante, l'encombrement sous des abris étroits.[g] According to M. Baudens, medical inspector of the French army, 'les 'causes du typhus sont connues à tel point, qu'on pourrait faire 'naître et cesser à volonté l'influence typhique;' and these causes were: 'la concentration et l'accumulation amenés par la rigueur 'de l'hiver. Les soldats entassés dans leurs tentes, hermétique-'ment fermées, subirent fatalement l'empoisonment, par le 'miasme organique.'[h] Lastly, observed Jacquot: 'Pas une con-'testation ne s'est élevée au sujet de la cause du typhus; les faits 'sont clairs et parlants; le typhus spontané est dû aux miasmes 'humains qui s'exhalent au milieu de l'agglomeration, de l'encom-'brement, etc. On peut faire naître le typhus à volonté, pour ainsi dire.'[i] Nor did the Russian surgeons think differently. M. Alferieff, professor of pathology, at Kiev, who was sent to investigate the sanitary state of the Russian army, reported: 'As the result of 'this over-crowding, the typhus appeared. In all cases, over-'crowding must be recognized, if not as the unique, yet as the 'essential and most active cause, of the epidemic.'[k]

e. Hospital Fever.

Typhus has often been observed to originate in over-crowded and badly ventilated hospitals. 'Hospital Fever' was the name given to the disease by Sir John Pringle, 'The hospitals of an army' said Pringle, 'when crowded with sick, or at any time when the air is 'confined, produce a fever of a malignant kind, and often mortal. 'I have observed the same arise in foul and crowded barracks.'[l] From his account of the symptoms, there is no doubt that he referred to typhus. In the present state of our civil hospitals, such occurrences are rare; but it is important to notice that typhus may originate in overcrowded hospitals, in countries where the disease is not endemic. For example, after the capture of Rome in 1849, typhus broke out in the crowded hospitals of the French troops.[m]

[f] Armand, 1858, p. 406. [g] Scrive, 1857, p. 409. [h] Baudens, 1858, pp. 230-2.
[i] Jacquot, 1858, pp. 64, 305. [k] Alferieff, 1856, p. 126.
[l] Pringle, 1752, p. 291. [m] Jacquot, 1858, p. 72.

f. A Contagious Fever in the Lower Animals.

During the American war, sheep and pigs were transported in large numbers, from England to America, and it was often found that a contagious fever broke out among them, on board the ships in which they were crowded.[n] Hildenbrand mentions similar facts, with regard to cattle;[o] Blane, with regard to dogs;[p] and Fergusson, in reference to sheep.[q] Professor Gamgee, Principal of the Veterinary College at Edinburgh, informs me that a contagious disease, in every respect resembling true typhus, is often generated in the lower animals, by overcrowding.

With such evidence, it is surprising that there is now any difference of opinion as to the spontaneous development of typhus. Down to the commencement of the present century, no doubt existed on the matter. Long ago, Lord Bacon remarked: 'The 'most pernicious infection, next to the plague, is the smell of the 'jail, where the prisoners have been long and close and nastily 'kept, wherein we have had experience twice or thrice, when both 'the judges that sat upon the bench, and numbers who attended 'the business, sickened upon it and died.'[r] All our great physicians of the past, Huxham, Pringle, Cullen, D. Monro, Blane, Stanger, Bateman, etc., re-echoed the opinion of England's first Lord Chancellor, and most emphatically declared that the poison of typhus often originated *de novo* under the circumstances above specified. Their opinions cannot be summed up better than in the quaint, but expressive language of William Grant, as contained in his essay on the 'Pestilential Fever of Sydenham:' 'If any person will take the trouble to stand in the sun and look 'at his own shadow on a white-plastered wall, he will easily 'perceive that his whole body is a smoking dunghill, with a 'vapour exhaling from every part of it. This vapour is subtle, 'acrid, and offensive to the smell; if retained in the body it 'becomes morbid; but if re-absorbed, highly deleterious. If a 'number of persons, therefore, are long confined in any close place, 'not properly ventilated, so as to inspire and swallow with their 'spittle, the vapours of each other, they must soon feel its bad 'effects. Bad provisions and gloomy thoughts will add to their 'misery, and soon breed the *seminium* of a pestilential fever, 'dangerous not only to themselves, but also to every person who

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[n] FORDYCE, 1803. 1st. Dis. p. 112.
[o] HILDENBRAND, 1811, p. 300.
[p] BLANE, 1789. 3rd. ed. p. 229.
[q] FERGUSSON, 1846, p. 166.
[r] *In Sylva Sylvarum.* Cent, x. No. 914.

'visits them, or even communicates with them at second hand. 'Hence it is so frequently bred in jails, hospitals, ships, camps, 'and besieged towns. A *seminium* once produced, is easily spread 'by contagion.'[s]

In 1811, appeared Bancroft's essay on the Yellow Fever, Typhus, etc.,[t] in which the author endeavoured to combat the opinion prevalent at the time, and to show that typhus and every other contagious disease invariably arises from 'the very same species of 'contagion, previously and in like manner elaborated in another 'body.' Never has any work effected a greater revolution in professional opinion in this country. The doctrine of Bancroft was generally adopted, without investigation of the facts upon which it was founded. A careful perusal of Bancroft's work, with an investigation of many of the facts to which he appeals, have convinced me that his doctrines are untenable, and that they have tended greatly to retard the progress of sanitary science. The evidence which he adduced with regard to typhus was entirely negative, such as the non-production of typhus from over-crowding among the Greenlanders and Esquimaux, in slave ships,[u] in the Black Hole of Calcutta,[x] and in Continental prisons.[y] He did not mention a single fact to show that in his own country, human beings were over-crowded, without typhus appearing among them; but he exhibited considerable ingenuity, in order to account for the circumstances of the 'black assizes.' The disease at the Old Bailey in 1750, he maintained to be not typhus, but to be due to the continued stream of cold air from the open window in the court!

But although many English physicians still adhere to the doctrine of Bancroft, the events of the Crimean war have opened the eyes of our brethren on the Continent, who, with few exceptions, regard the spontaneous generation of typhus as an unassailable fact. The opponents of this view, are forced into the position of admitting, that the typhus-poison is in every place, ready to manifest itself under the conditions above specified, and under no other.

There are certain conditions, more or less essential to the production of the typhus-poison from over-crowding, which must not be lost sight of.

1. *Defective Ventilation* must co-exist with over-crowding. Over-

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[s] GRANT, 1775, p. 7. [t] BANCROFT, 1811. [u] See p. 113 of this work.
[x] Ibid. p. 113. [y] Ibid. pp. 105-7.

crowding must always be considered in relation to the amount of ventilation. The degree of crowding, which, with defective ventilation, or with none at all, would generate typhus, would be harmless if the surrounding atmosphere were repeatedly renewed.

2. *Personal Squalor, and Filthy Apparel* saturated with cutaneous exhalations, greatly aggravate the bad effects of over-crowding, and may be necessary for the production of the poison. Hence, perhaps, the frequency of typhus among the lower classes of Irish, and possibly one cause of the reported exemption from contagious fever of the naked negroes in slave ships.[z]

3. *A deteriorated state of the constitution,* such as results from protracted starvation, scurvy, and other debilitating causes, favours the development of the typhus poison, and may possibly be an essential condition.[a] During periods of famine or local scarcity, overcrowding is more likely to produce typhus than at other times.

The alleged exemption of the Laplanders and Esquimaux from typhus, notwithstanding the badly ventilated state of their dwellings, may be due to the extreme cold of the climate, or to their being well nourished. It has not been shown, that typhus does not prevail among them during famine.

4. *A considerable time is necessary for the production of the poison.* There are many examples of a number of men being crowded in such a confined space, that some have died within a few hours, and yet no contagious fever has appeared among the survivors. That most commonly referred to, is the tragedy of the 'Black Hole of 'Calcutta,' which occurred in the night of June 20th, 1756. 'Figure to yourself,' said Governor Holwell, the historian, and one of the survivors of the event, 'if possible, the situation of 146 'wretches, exhausted by continual fatigue and action, thus 'crammed together in a cube of eighteen feet, in a close, sultry 'night in Bengal, shut up to the eastward and southward (the 'only quarters from whence air could reach us) by dead walls, 'and by a wall and door to the north, open only to the westward 'by two windows, strongly barred with iron, from which we could 'receive scarce any the least circulation of fresh air.' Of the 146 persons shut up by the orders of Surajut Dowla at eight in the evening, 123 were corpses at six next morning; 23 only came out alive. The symptoms from which they all suffered, were excessive perspiration, followed by violent thirst (which Holwell relieved by sucking the perspiration from his own shirt-

[z] Bancroft, 1811, p. 127; Trotter, 1803, i. 185; Fergusson, 1846, p. 176.
[a] Hildenbrand, 1811, p. 301; Jacquot, 1858, p. 70.

sleeves), great dyspnœa, palpitations, delirium, and insensibility. All who survived were seized with a 'putrid fever,' which was characterized by an eruption of boils, but which was in no case fatal, and was not apparently typhus.[b]

In this, and in all like cases,[c] death has resulted from asphyxia, and the non-production of the typhus poison cannot justly be adduced as an argument against the possibility of its spontaneous origin. There was not sufficient time for its development.

5. *Over-crowding favours the spread and development of other diseases besides typhus*, such as cholera, trismus nascentium, and scrofulous affections.[d]

The connection between dysentery and typhus is of great interest. Sir Gilbert Blane, Dr. Copland, and others, have tried to show that these two diseases are sometimes vicarious, dysentery in the black, taking the place of typhus in the white, man.[e] The disease which carries off the wretched Africans in the crowded holds of slave ships is not typhus, but dysentery; and yet the African is known not to be exempt from typhus. The Arab crew of the Scheah Gehaad suffered, not from typhus, but from dysentery, though they communicated typhus to others (see page 108). Again, in temperate climates, typhus is sometimes complicated with dysentery, while epidemics of the two diseases often co-exist, and apparently originate from similar causes, among persons of the same race. This subject merits investigation.[f]

It is perhaps premature or rash to hazard a conjecture as to the actual nature of the typhus-poison; but there are some grounds for supposing with Liebig, Simon, Scherer,[g] Viale and Latini,[h] and Richardson,[i] that it is a compound of ammonia. It is long since Winter[k] expressed the opinion, that the presence of ammonia in the blood accounted for the phenomena of typhus; and Richardson has shown that ammonia, introduced artificially into the blood, 'produces what may be unhesitatingly considered typhoid symp-'toms.' In severe cases of typhus, not only is there reason to believe that the blood is ammoniacal, but the exhalations from the lungs and

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[b] Holwell, 1758.

[c] For similar occurrences to that of the Black Hole of Calcutta, see account of the Irish steamer 'Londonderry' (Carpenter's *Princip. of Hum. Phys.*, 5th ed. p. 300), the tragedy of Ujnala (*The Crisis in the Punjaub, by* F. Cooper, C. S. Lond., 1858, p. 162); Wells, *On the Health of Seamen*, 2nd ed. p. 17; and Barrallier, 1861, p. 31.

[d] See Carpenter's *Princ. of Hum. Phys.*, 5th ed. p. 301. [e] Duncan, 1862.

[f] See Discussion at Epidemiological Society. *Brit. Med. Journ.*, Aug. 10, 1861.

[g] Quoted by Haller, 1853; Hudson, 1841, p. 3. [h] Viale and Latini, 1854.

[i] Richardson, 1858. [k] Lehmann's *Phys. Chem.* (Day's ed.) i. 453.

skin and the discharges from the bowels contain a large amount of ammonia. It is a common observation, that a pungent ammoniacal odour is given off by the skin and lungs in typhus, while the presence of a large quantity of ammonia in the breath admits of actual demonstration.[1] Now, it has been ascertained by Gerhard[m] and others, that the cases in which this odour is strongest, communicate typhus most readily to persons in health. Again, in many of those cases, where the symptoms of typhus have supervened immediately on exposure to the source of contagion, and where we may suppose the poison to have been unusually concentrated, the affected persons have been conscious at the time of exposure, of a most disagreeable odour, which has been described as pungent and ammoniacal.[n]

This view as to the nature of the typhus poison is not incompatible with its spontaneous origin. The experiments of Berzelius and other chemists showed, that ammonia was contained in the cutaneous exhalations in health;[o] and the researches of Marchand, the Rev. J. B. Reade,[p] Viale and Latini,[q] Reuling,[r] and Dr. Richardson, have shown that minute traces of ammonia are constantly exhaled in healthy respiration. Although the quantity be much smaller than in typhus, it is probable that, when a large number of human beings are crowded into a small unventilated space, the ammoniacal exhalations are increased and concentrated, and, by their putrefaction, generate the poison of typhus. The first effects of over-crowding with no ventilation, is to cause the respiration of an atmosphere charged with carbonic acid; but it has been shown by experiment, that even a small per centage of carbonic acid, in the respired air, is sufficient to cause a serious diminution in the amount of carbonic acid thrown off, and of oxygen absorbed. 'It 'follows,' says Dr. Carpenter, 'that those oxidating processes 'which minister to the elimination of effete matter from the 'system must be imperfectly performed, and that an accumulation 'of substances tending to putrescence must take place in the 'blood. Hence there will probably be a considerable increase in 'the amount of such matters in the pulmonary and cutaneous exha-'lation;'[s] and the unrenewed air will become charged, not only with carbonic acid, but also with organic matters in a state of decomposition, of which the chief product is ammonia.

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[1] See under head 'of Respiration in Typhus.'

[m] GERHARD, 1837, xx. 298.

[n] See p. 89, and MARSH, 1827.

[o] RICHARDSON, 1858, p. 358.

[p] *Gardener's Chronicle*, 1847.

[q] VIALE and LATINI, 1854.

[r] LEHMANN'S *Phys. Chem.* (DAY'S Transl.) iii. 559.

[s] *Princ. of Hum. Phys.* (5th ed.) p. 301.

Again, the normal quantity of ammonia contained in the breath is exceeded in other diseases, besides typhus. What those diseases are, remains to be shown; but in several, as for example, uræmia from degeneration of the kidneys, I have rarely failed to find ammonia in considerable quantity in the breath. This circumstance may possibly account for the spontaneous development of typhus in unventilated hospitals, under circumstances of crowding insufficient to generate it elsewhere, and likewise for the observation that a deteriorated state of the constitution predisposes to the development of the typhus-poison.

From the present state of our knowledge, it seems not unreasonable to conclude, that the disagreeable odour of the cutaneous and pulmonary exhalations of typhus patients, as well as the offensive smell generated by over-crowding, are due to some unknown compound of ammonia, which is the typhus-poison. But, leaving the realms of conjecture, the etiology of typhus may be summed up thus:

1. Typhus is due to a specific poison.
2. This poison is communicated from the sick to the healthy, through the atmosphere or by fomites, but is rendered inert by free ventilation.
3. The poison is also generated *de novo*, by over-crowding and bad ventilation.
4. The great predisposing cause of typhus is defective nutrition.

Sect. VI. Symptoms of Typhus.

IT may be advantageous to give, in the first place, the connected clinical history of a typical case, with reports of a few cases in illustration, and then to proceed to an analysis of the individual symptoms.

a. Clinical Description.

The advent of typhus is, in most cases, somewhat sudden. Occasionally, it is preceded by one or more days of slight indisposition, characterized by lassitude, vertigo, slight headache and loss of appetite, but not such as to incapacitate the patient from following his ordinary employment. With, or oftener without, these premonitory symptoms, the patient is seized with slight rigors or chilliness, followed by lassitude and disinclination for exertion, frontal headache, pain in the back, pains like those from bruises in the limbs, especially in the thighs, loss of appetite, and often, for a day or two, irregular chills and slight perspirations. For two or three days, although the skin feels hot and burning,

the patient complains of chilliness, and sits close to the fire. The tongue is large, pale, and covered, first with a white fur, and afterwards with a yellowish brown coat; the appetite is gone; the taste is perverted, and there is more or less thirst; the patient fancies different drinks, but he soon loathes all except cold water. Occasionally there is nausea, but rarely vomiting; the abdomen is free from pain or tenderness; the bowels are constipated; and the urine is rather scanty, high-coloured, and dense. The pulse varies from 80 to 120; it is often full, but almost always compressible; only in rare cases, has it any firmness. The respirations are slightly accelerated, and sometimes there is a feeling of oppression at the chest, with slight cough and mucous expectoration and some sonorous râles. The face is flushed and dusky; the edges of the eyelids are tumefied, the conjunctivæ are injected; and the eyes water. The expression at first betokens languor and weariness, but soon becomes dull, heavy and stupid. From the first, there is vertigo, tinnitus aurium, restlessness, and often total loss of sleep; but frequently the patient declares that he has not slept, and yet the attendants have watched him sleeping for hours. The sleep is disturbed by painful dreams and sudden starts, and, after three or four nights, there is talking in the sleep, with slight delirium between sleeping and waking. When awake, the patient is still conscious, though perhaps somewhat confused in memory and intellect. With all this there is early and rapidly increasing muscular prostration; the gait is tottering, the hand, extended to the physician, shakes, and there may be trembling of the protruded tongue; soon there is an intolerable sensation of complete exhaustion, so that about the third day, the patient is compelled to keep his bed.

Between the fourth and the seventh days, usually on the fourth or fifth, an eruption makes its appearance on the skin. It is composed of numerous spots of irregular form, varying in diameter from three or four lines to a mere speck, which are either isolated or grouped together in patches presenting a serpigenous or very irregular outline, and often closely resembling the eruption of Measles. At first, these spots are of a dirty-pink or florid colour, and very slightly elevated above the skin, and they disappear upon pressure; but, after the first or second day, they usually become darker and more dingy, they resemble reddish-brown stains, are no longer elevated above the skin, and do not disappear, but only become a little paler, on pressure. They have no defined margin, but merge insensibly into the colour of the surrounding skin. These spots usually come out first over the

abdomen, and thence they spread to the chest, back, shoulders, thighs and arms; in some cases they are first seen on the backs of the hands; they are most common on the trunk and arms, and are rarely observed on the neck or face. Along with these superficial spots, there are others which are paler, less distinct, and which, from their apparent situation, beneath the cuticle, have been designated 'subcuticular.' When abundant, this subcuticular rash imparts to the skin a mottled or marbled aspect, which contrasts with the darker more defined spots before described, although sometimes the two appear to pass into one another. The eruption of typhus varies greatly in its appearance, according to the relative abundance of the mottling and more distinct spots. Sometimes both are plentiful; sometimes there are only a few of the more distinct spots; and at other times, there is nothing but a faint subcuticular mottling, which is apt to be overlooked. Its appearance also varies according to the degree of isolation or confluence of the distinct spots. The spots and mottling together constitute an eruption which Jenner has described as the 'Mulberry rash' of typhus, but which other writers have designated measly, morbilliform, or rubeoloid. (See Plate I., and p. 127).

This is the history of typhus during the first six or seven days of the disease.

About the end of the first week, the headache ceases, and delirium supervenes. This delirium varies in character. Occasionally it is at first acute; the patient shouts, talks incoherently, and is more or less violent; if not restrained, he will get up and walk about the room, or even throw himself from an open window. This violent state is usually followed by great collapse, or the noisy condition passes into low, muttering delirium. More commonly, the delirium is never acute, even at first. With either form there is usually sleeplessness; and when spoken to, the patient is rendered more excitable. The countenance becomes more dusky, the conjunctivæ more injected, and the expression more dull and stupid, while the prostration hourly increases. The symptoms of nervous excitement are usually most marked towards evening, and in the night-time; the prostration is greatest in the morning. At the same time, the tongue becomes dry, brown, and rough, along the centre, and is tremulous; sordes collect upon the teeth and lips; constipation continues. The pulse varies from 100 to 120, and may be full and soft, but is oftener small and weak; the respiratory movements vary from twenty to thirty, and the breath is fetid. The skin is cooler than during the first week; it is dry, or slightly clammy, and gives off a peculiar odour. The eruption

assumes a darker shade, and about the middle of the second week, true petechiæ, of a purple or bluish tint, may appear in the centre of many of the spots, these petechiæ at their edges gradually merging into the reddish-brown hue of the primary spots. (See Plate II).

After three or four days, the symptoms of nervous excitement are succeeded by more or less nervous depression and stupor. At first, the stupor and delirium alternate, the latter being most marked in the night-time. The prostration is extreme: the patient lies on his back, moaning, muttering incoherently, or still and motionless, with a tendency to sink to the bottom of the bed. He is quite unable to raise himself, or even to turn on his side, and is with difficulty roused, and is utterly indifferent to surrounding objects and persons. Tremors, subsultus, and picking of the bed-clothes may be observed. The expression is stupid and vacant; the conjunctivæ are injected, the eyelids, for the most part, closed, and the pupils often contracted. Deafness is not uncommon. If spoken to loudly, the patient opens his eyes, and stares vacantly at those about him, and when told to put out his tongue, he opens his mouth and leaves it open, until desired to close it. These are all the signs of consciousness exhibited; and even they may be absent. But all this time the mind is far from inactive; the imagination conjures up the most frightful fancies, to which implicit belief is attached, and of which a distinct recollection may remain after recovery. The ideas often revolve on previous events of the patient's life. He believes himself persecuted and tormented by his attendants and dearest relatives; he compresses years into hours, and in a few hours imagines that he has lived a life-time. They who have passed through these mental sufferings can alone imagine their intensity. The teeth and lips are now covered with sordes; the tongue is hard and dry, dark brown or black, contracted into a ball, tremulous, and protruded with difficulty or not at all. The abdomen is flaccid, or sometimes tympanitic; the bowels are still confined, or one or two slightly relaxed motions are passed daily in bed. The urine is more copious, but paler, and of low specific gravity, and is passed involuntarily, or retained, so as to necessitate recourse to the catheter. The skin is cooler than before, and sometimes moist; the number of spots presenting a petechial character increases. The parts subjected to pressure, and particularly the skin over the sacrum, become red and tender, and are liable to slough. The pulse is frequent (120 to 140), small, and weak, and not unfrequently intermittent, irregular, or scarcely perceptible; the cardiac impulse and systolic sound of the heart are diminished in intensity, or absent.

In this state, the patient may continue for many hours, or several days, with life trembling in the balance, until, at last, the stupor passes into profound and fatal coma; or sudden engorgement of the lungs, with asphyxia, supervenes; or the pulse becomes imperceptible, the surface cold, livid, and bathed with copious sweat, and death ensues usually without any return to consciousness, but likewise without stertor, and from syncope rather than coma.

But, on or about the fourteenth day, there is often a more or less sudden amendment. The patient falls into a quiet sleep, which lasts for several hours, and from which he awakes another man. At first he is bewildered and confused, and wonders where he is; but he recognises his attendants and friends; and he is now conscious of his extreme debility. His extremities retain their sensibility; but when he attempts to move them, they seem at first as if separated from the body. The pulse has fallen, and is of better strength; the skin is moist; the tongue is clean and moist at the edges; there is a slight desire for food, and the delirium has ceased. These symptoms of improvement are often accompanied by slight perspiration, diarrhœa, or a deposit of lithates in the urine. After two or three days, the tongue becomes altogether clean, the appetite is insatiable, the pulse has fallen to its normal standard, or is unusually slow, and the strength is rapidly regained. No permanent mischief is left behind.

Such is the clinical history of a typical case of uncomplicated typhus. But the disease presents great varieties, according to its severity, and the relative preponderance of the adynamic (cardiac) or ataxic (cerebral) symptoms. In mild cases, the tongue may be never dry and brown, the pulse may never reach 100, the rash may never become petechial, and slight confusion of the memory and intellect, and disturbed sleep, may be the only symptoms of cerebral derangement. The course and characters of the disease are also very apt to be modified by the local complications, to be hereafter mentioned.

B. Illustrative Cases.

Case I.

Typhus Fever, with Symptoms of moderate severity.—Commencement of Convalescence on the fourteenth day.

Charles S——, aged 23, admitted into the London Fever Hospital, April 10th, 1862. Six days before admission was seized with chilliness, loss of appetite, and pains in the limbs, followed by severe frontal headache. These symptoms got worse. On the third day of his illness, he was obliged to take to his bed.

On admission,—pulse 104; tongue moist and thickly coated; much thirst; no appetite; bowels confined. Face generally flushed; conjunctivæ injected; has a stupid, heavy expression. Still complains much of head-ache and pains in the limbs, but they are less severe than formerly. A copious typhus rash over trunk and arms, some of the spots disappearing on pressure; but others persistent. Skin hot and dry. Was ordered a mixture containing nitro-muriatic acid and nitre,[s] also castor oil, four ounces of wine, beef-tea, and milk. Tepid sponging.

April 13th (ninth day). Pulse 120. Head-ache ceased, and patient is now free from all pain; but countenance is more heavy; is rather confused when spoken to, and somewhat deaf. Restless in the night, but no delirium. The eruption is less copious, but what remains is darker, and persistent on pressure. Tongue dry, rough, and brown along the centre; bowels were moved twice by the oil, and once to-day. Urine of a clear amber colour; sp. gr. 1016, acid, and free from albumen, but contains a slight mucous cloud composed of vesical epithelium.

April 16th (twelfth day). Pulse 120; is more prostrate, but otherwise much in the same state as on the 13th. No delirium, but is more deaf and stupid. Urine has been examined daily, and is still free from albumen, but yesterday it deposited a quantity of pale lithates. Four ounces of brandy substituted for wine.

April 17th (thirteenth day). Pulse 120; tongue dry and brown; bowels open once daily. The eruption has not got darker since the 13th. Still deaf, confused, and stupid; but has no delirium, and sleeps at intervals. Urine of a clear amber colour; sp. gr. 1012, free from lithates, but contains a slight trace of albumen.

April 18th (fourteenth day). Much better on awaking this morning. Pulse 90, and of better strength; less stupid; tongue moist at the edges, and rash disappearing. Urine of a dark amber colour, and free from both albumen and lithates.

Was ordered to have an egg, and a mixture containing bark and mineral acids.

April 20th (sixteenth day). Pulse 72. Tongue clean and moist; bowels open; is very hungry; rash gone. Urine free from albumen and lithates, both to day and yesterday. Was ordered meat and porter.

The patient rapidly regained his strength, and left the Hospital on May 2nd. The urine was examined daily till April 26th, but continued free from albumen and lithates.

Case II.

Typhus Fever, with severe Cerebral Symptoms.—Commencement of Convalescence on the fourteenth day.

James C——, admitted into the London Fever Hospital, April 12th, 1862. Was seized six days before admission with slight rigors and chilli-

[s] The reader is referred to the Section on Treatment, for the Prescriptions in this and the following cases.

ness, followed by severe head-ache and pains in the back, loss of appetite, and restless nights. Was obliged, from weakness, to take to bed on the second day of his illness.

On admission,—pulse 90, full and not very weak. Still much head-ache and pain in the back, but no delirium. Tongue moist and furred; much thirst; bowels open from medicine; skin hot and dry. A well marked typhus eruption, consisting of mottling and distinct reddish-brown spots, not disappearing on pressure, over chest and abdomen. Was ordered a mixture every three hours, containing nitro-hydrochloric acid and nitre; also beef tea and milk. The body to be sponged twice daily with a solution of Condy's fluid.

April 13th (eighth day). No delirium, but is very confused, and expression stupid. Head-ache much relieved. Urine slightly turbid from lithates, sp. gr. 1024, acid, free from albumen, and containing scarcely a trace of chlorides. No cough, and physical signs of chest normal.

On the night of April 13th, he became very delirious, and could with difficulty be kept in bed. This delirium continued during the two following nights; in the day-time he was quieter, and answered when spoken to. On the 14th, his tongue was dry and brown at the base and along the centre. On the nights of the 14th and 15th, sleep was obtained by means of the antimony (gr. $\frac{1}{16}$) and morphia (gr. $\frac{1}{6}$) draught. The dose was repeated every hour until the patient slept. Two doses were sufficient. On the 14th he was ordered four ounces of wine, for which, on the 15th, four ounces of brandy were substituted.

April 16th (eleventh day). More prostrate. Pulse 112, and feeble; takes notice when spoken to, but is scarcely conscious, and very deaf. Much low muttering delirium, and still now and then makes attempts to get out of bed. Face flushed and dusky; conjunctivæ injected, and pupils contracted. Eruption copious and darker, and many of the spots distinctly petechial. Tongue dry and brown, and scarcely protruded; sordes on teeth. Much ammonia in the breath (see p. 139). Urine has been examined daily for albumen and chlorides, but has contained none of the former, and scarcely a trace of the latter, although the patient was made to take two drachms of common salt on the morning of the 14th, and again on the 15th. The brandy was increased to eight ounces, and a mixture containing sulphuric acid, sulphuric ether, and quinine, was substituted for that used on admission, which had been omitted on April 13th, so as not to interfere with the observations on the urine. On the 18th, the brandy was increased to twelve ounces.

April 19th (fourteenth day). Much more prostrate; lies on back, and can scarcely move in bed. Pulse 120, very feeble and irregular. Much tremors and low muttering delirium; patient is quite unconscious; pupils extremely small. Yesterday the stools and urine were passed involuntarily: to-day the bladder was enormously distended, and the urine had to be drawn off by catheter. Tongue dry, brown, and crusted. Copious typhus petechial rash; slight cough. Urine still free from albumen.

Was ordered to have a large mustard poultice to the chest and epigastrium, and to continue the brandy and the mixture ordered on the 16th.

The same evening (fourteenth day) the symptoms began to improve; and on the following day, the pulse had fallen to 100, the tongue was moist at the edges, the rash was fading, the skin moist, and the patient took more notice when spoken to.

April 21st (sixteenth day). Is still very prostrate, deaf, and a little confused; but pulse 72, can pass water freely, and motions and urine are no longer involuntary; tongue moist.

Ordered mineral acids and bark, and the brandy to be reduced to six ounces.

On April 22nd and two following days, the pulse did not exceed 46, but all the other symptoms continued to improve.

On the 25th the pulse was 72, the tongue clean and moist, the consciousness was quite restored, and the patient was free from all complaint except great weakness. The brandy was discontinued, and meat and porter were ordered. Convalescence progressed rapidly, and on May 5th the patient was discharged from the Hospital, well.

Case III.

Typhus Fever, with great Prostration and Cerebral Symptoms.—Convalescence at the end of the second week.

John B——, aged 54, was admitted into the London Fever Hospital, April 12th, 1862. On admission, the patient was confused in his mind, and said that he had been ill for five weeks, although the real duration of his illness was only eight days, but in other matters he answered correctly. Pulse 108, feeble, and intermitted regularly after every third beat. Impulse of heart very feeble, but first sound audible. Had a stupid expression of countenance, and conjunctivæ were much injected, but was free from all pain. A copious typhus-rash over trunk and arms, all of the spots dark reddish-brown, and persistent on pressure. Tongue dry and brown along centre, but moist at the edges; one motion daily; urine clear, acid, sp. gr. 1014, and contained a considerable amount of albumen.

Was ordered a mixture containing nitro-hydrochloric acid and nitre, also beef-tea, milk, and four ounces of brandy, and a sinapism to the loins.

April 14th (eleventh day). Pulse 128, feeble, and still intermittent. Much tremor, and two involuntary stools. Still confused, and has been occasionally delirious; pupils contracted; urine clear, and acid; sp. gr. 1014; quantity of albumen less.

The brandy was increased to eight ounces.

During the whole of April 15th the patient continued in the same state, but was so weak that he could not move in bed. The urine still contained much albumen.

On the morning of the 16th (thirteenth day), there was a marked improvement. The pulse had fallen to 96, and was regular and of better strength. The countenence had improved, and the delirium had ceased.

The motions were no longer passed in bed. The tongue was moist and cleaning; the rash was fading, and there was some appetite. The quantity of albumen had greatly diminished, and on the following day it quite disappeared, Although the urine was examined daily, no deposit of lithates took place. For about a week, the patient remained very weak, but after this he rapidly regained his strength.

Case IV.

Typhus Fever with severe Cerebral Symptoms, Tremors, Subsultus, Convulsions and Coma.—Death on the thirteenth day.—Autopsy: Hyperæmia of Internal Organs. Increase of Cerebral Serosity and Softening of the Heart.

Thomas M——, aged 36, admitted into the London Fever Hospital, May 12th, 1862. Out of employment for many weeks. Was taken ill six days before admission with rigors and loss of appetite. Although he felt very weak, he continued going about until May 11th.

On admission, pulse 96, and weak. Tongue dry and brown along the centre; bowels open from medicine. A well-marked typhus-eruption, the spots persistent on pressure, on chest and abdomen. Eyes injected; face flushed; answers correctly, but is rather excited; and says he is afraid to go to sleep for fear of something happening to him. Has had much pain in limbs and headache, but the pains have almost ceased.

Was ordered beef-tea and milk, four ounces of wine, and a mixture containing sulphuric acid, sulphuric ether, and quinine.

April 14th (ninth day). Is more prostrate. Hands and tongue tremulous. Is stupid and confused, and occasionally delirious. Pulse 120. Tongue dry and brown. Urine passed in bed.

Four ounces of brandy were ordered.

On the evening of the 14th, he had a slight convulsive fit, with foaming at the mouth, lasting for a quarter of an hour. After this he became drowsy and unconscious, and scarcely took notice when spoken to; the tremors increased and there was also subsultus; the abdomen was tympanitic, and the motions and urine were passed involuntarily. The urine was ascertained to contain a considerable quantity of albumen.

The brandy was increased to ten ounces. A strong infusion of coffee was ordered to be taken every four hours. The bowels were freely moved and sinapisms were applied to the loins.

The patient, however, became weaker; on the 17th, he was comatose, and he remained in this state until his death on the 18th (thirteenth day).

Autopsy. Twenty-four hours after death.

Post-mortem rigidity slight. Typhus-spots distinct.

Sinuses of dura mater filled with dark fluid blood. Moderate vascularity of pia mater. A considerable amount of sub-arachnoid serosity, sufficient at some places to elevate the membrane above the surface of the convolutions. Three drachms of fluid in each of the lateral ventricles.

Brain-substance of normal colour and consistence, and not abnormally vascular.

Muscular substance of heart, soft, friable, and pale, and the muscular fibres at many places in a state of fatty degeneration. Right cavities filled with dark fluid blood. Moderate hypostatic congestion of both lungs, which were otherwise healthy.

Intestines healthy. Not the slightest vascularity or elevation of Peyer's patches, or of the solitary glands of the ileum. Liver moderately hyperæmic. Spleen nine ounces, soft and diffluent. Kidneys of normal size, surfaces smooth; renal tissue loaded with blood; and uriniferous tubes gorged with epithelium.

Case V.

Typhus, with symptoms of moderate severity. About the fifteenth day, sudden rise of pulse, profuse sweating, and rapid sinking. Autopsy:—Softening of Heart, Hyperæmia of the Internal Organs, &c.

Alexander R——, aged 23, admitted into London Fever Hospital November 18, 1857. No information could be obtained with regard to his history, except that he had been ill for ten or eleven days, and in bed for a week.

On admission, pulse 80, and small. Lies on his back, and has a heavy confused expression, but answers well. Does not complain of pain anywhere. Tongue moist and covered with a yellow fur. Bowels confined. An indistinct typhus-mottling over chest and abdomen. Ordered six ounces of wine, beef-tea, and carbonate of ammonia.

November 19th (thirteenth day). Pulse 80. Slept little, and was very delirious during the night, and is scarcely conscious. Tongue still moist. Bowels opened by oil.

Continued very much in the same state, the pulse never exceeding 84, until the morning of November 21st (fifteenth day), when, about eleven a.m., he began to perspire profusely. The skin was cold, and the extremities and face livid. The pulse rose to 150, and was scarcely perceptible; the tongue became dry and brown; the respirations were quickened; but there was no cough, and no dulness could be made out in the chest. Brandy and diffusible stimulants were freely administered, and a blister was applied to the nape of the neck, but the patient continued to sink, and died at nine p.m.

On *post mortem* examination, the texture of the heart was found to be softened and pale; the blood was dark and fluid; the lungs were healthy, with the exception of slight hypostatic congestion. The liver and kidneys were very hyperæmic; the spleen weighed seven ounces, and was very soft. Peyer's patches and the solitary glands of the ileum were perfectly healthy. The pia mater was much injected; the arachnoid was slightly raised above the convolutions by serosity; and each of the lateral ventricles contained about three drachms of clear fluid. The brain-substance was healthy.

Case VI.

Typhus, with severe Cerebral Symptoms. Coma-Vigil and Death on the sixteenth day. Autopsy:—Great Hyperæmia of Internal Organs, Softening of Heart, and Hypostatic Condensation of Lungs.

James S——, aged 48, a cabman, was admitted into the London Fever Hospital March 18th, 1862. Had rigors on the 12th, and took to bed the same day, with frontal headache, giddiness, severe general pains, weakness, and loss of appetite.

March 19th (eighth day). Pulse 106, full but compressible. Slept tolerably well last night, and had no delirium; headache and general pains have almost ceased. Face dusky. Expression stupid. Conjunctivæ injected. Tongue dry and brown along the centre; bowels confined. Skin hot and dry; copious reddish-brown typhus-rash on trunk.

Was ordered four ounces of wine, beef tea, and milk, and a mixture containing mineral acids and nitre.

March 21st (tenth day), During the two last nights has slept little, and has been very restless, and occasionally delirious, attempting to get out of bed. Pulse 100, and feeble. Tongue moist, but thickly coated. Bowels opened by oil. Eruption darker, and many of the spots have become converted into petechiæ.

Was ordered six ounces of brandy, and a draught, to be repeated every hour at night, containing $\frac{1}{16}$th grain of antimony, and $\frac{1}{6}$th grain of morphia.

March 23rd (twelfth day). Slept during two last nights, after second dose of draught, but is much more prostrate, and scarcely conscious; pupils contracted. Much tremor, with occasional subsultus and floccitatio. The stools and urine passed involuntarily. Pulse 120, and feeble. Heart's impulse feeble, and first sound almost inaudible.

The brandy was increased to ten ounces, and a mixture was ordered every three hours, containing sulphuric acid, sulphuric ether, and quinine.

No improvement took place, and on the morning of the 26th (fifteenth day), the patient was much worse. Quite unconscious; eyes fixed and staring; pupils much dilated, and scarcely affected by light. Much subsultus and floccitatio; stools and urine passed involuntarily. Pulse 130; respirations 50; face livid; percussion and auscultation in front of lungs normal; first sound of heart inaudible. The patient remained in this state until his death, on the following morning.

Autopsy. Twenty hours after death.

Sinuses of dura mater, filled with dark fluid blood; pia mater much injected, and bloody points in brain-substance numerous. A slight amount of sub-arachnoid serosity; nearly an ounce of fluid at base. Brain-substance of normal consistence.

Texture of heart pale and soft, especially of left ventricle; right

cavities filled with dark fluid blood, and soft black coagulum. Each of the lungs weighed about thirty ounces. Posteriorly both lungs were solidified, so as to sink in water. The solid portion extended nearly two inches inwards from the surface, was separated by no distinct line of demarcation from the healthy lung, and exhibited no granular appearance on section.

Liver and kidneys very hyperæmic. Spleen seven ounces, soft and pulpy. Peyer's patches and the solitary glands exhibited no ulceration, increased vascularity, or elevation.

C. Analysis of Principal Symptoms.

a. The Physiogomy.

The physiognomy of a patient suffering from typhus (*facies typhosa*) is peculiar, and often of itself suffices to indicate the disease. From the first it is dull and heavy; and, as the disease advances, it becomes more oppressed, vacant, and bewildered, while the eyelids and mouth are kept half open. In cases where there is acute delirium, the countenance may be correspondingly wild and defiant. At no time does the countenance betray an expression of anxiety, for in few cases does the patient suffer acute pain, and rarely is he concerned as to the issue of the disease. The face is often flushed. The flushing is general over the entire face, and, though sometimes greatest on the prominences of the cheeks, it is never circumscribed. It is never pink; sometimes it is reddish or reddish-brown, but more commonly it is of a dusky, earthy, or leaden hue; in grave cases, it may be livid. If to the above features, be added the injected, suffused conjunctivæ, the dry brown tongue, and the sordid lips and teeth, the physiognomy of typhus is complete. As a rule, the extent to which the typhus physiognomy is developed, is in direct proportion to the severity of the case.

b. Morbid Phenomena referrible to the Skin.

1. *The Typhus Eruption.*—The general characters of the typhus-eruption have been already described (see page 117). According to its colour, the eruption may be said to pass through three stages, viz,:—1, Pale-dirty-pink, or florid; 2, reddish-brown; 3, livid, and petechial.* In the first stage, it disappears on pressure; in the second, it disappears in part only; in the last, it is

* The different appearances of the eruption are represented in Plates I & II.

not affected by pressure. The duration of the several stages varies, and the eruption may be arrested, so to speak, at any of the stages. As a rule, the third stage is observed as early as the second day after its appearance, and the petechial not until the end of the second week of the disease; but the eruption does not necessarily become reddish-brown, still less livid or petechial. Of 139 cases at Glasgow in 1838, Dr. Stewart found it, never more than "pale" in 34, "florid" in 25, "dark" in 48, "livid" in 15, and "petechial" in only 17. This proportion will, of course, vary at different times and places. According to my experience in London, the number of cases in which the eruption becomes reddish-brown, so as not to disappear on pressure, is much greater than stated by Dr. Stewart. On the other hand, the eruption is occasionally reddish-brown, livid, or petechial, almost from the first.

The spots situated on the dependent parts of the body are always the darkest; and here they are sometimes distinct, while elsewhere they are scarcely visible. Hence, in doubtful cases, the back ought always to be examined.

The quantity of the eruption, its depth of colour, and the earliness with which it becomes livid or petechial, are in a direct ratio to the severity of the case. Rasori,[t] Henderson,[u] and Stewart[x] have shown that the duration and severity of the disease are in proportion to the abundance and darkness of the eruption, and that convalescence is more protracted when it is abundant than when it is scanty. Of 59 cases noted by Dr. Stewart, with a light-coloured eruption, 5 died, or only 1 in $11\frac{4}{5}$; but of 80, with a dark eruption, 21 died, or 1 in $3\frac{4}{5}$.[y] The ominous character of the livid and petechial eruptions has been mentioned by all writers from the earliest times. (See pages 21, 23, 27). Cases without any eruption are mostly mild.

Much discussion has taken place respecting the title to rank among the exanthemata, conferred upon typhus by the eruption.[z] On its first appearance, the eruption is undoubtedly a true exanthem, due to hyperæmia of the cutaneous capillaries. It is then of a pinkish or florid hue, disappears on pressure, and may be slightly elevated above the surface. The whole or part of the eruption may never pass beyond this state, and then, if death

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[t] Rasori, 1813, p. 15. [u] Henderson, 1839. [x] Stewart. 1840, p. 325. [y] Ib. p. 323.

[z] Hildenbrand, (1811), Roupell (1831), and Peebles (1835), all maintained the right of typhus to rank with the exanthemata. This view was opposed by West (1840) and others.

occur, no traces of spots are found on the dead body. But in most cases, sooner or later, an escape of blood-pigment into the cutis is substituted for the hyperæmia; the spots become darker, are no longer elevated, and do not disappear on pressure. The colour will vary according to the amount of pigment thrown out; if it be small, the colour is reddish-brown; if large, the spots are livid or petechial. The spots now persist after death, and, on examining microscopically thin sections of the skin made through them, the colour is found to be due to an infiltration of dissolved hæmatine into the tissue of the cutis. In the reddish-brown spots, the tinging is limited to the surface of the true skin; but in the darker forms it extends through the entire thickness of the cutis, and sometimes even into the subcutaneous areolar tissue. The changes described may take place in a portion of the spots only, the others remaining pale or florid and non-persistent on pressure, or disappearing entirely. The subcuticular mottling also often disappears after a few days, while the spots continue to get darker. Hence, the eruption of typhus is often pale and confluent in its early stage, darker and more spotted in the advanced.

Since the days of Nicholas Massa[a] and Sennertus,[b] typhus has often been designated 'Petechial Fever'; but the term *petechiæ* is used in very different significations, and hence has arisen great confusion. Rochoux restricted the term *petechiæ* to the eruption of typhus, although he regarded it as a true exanthem, and not due to local hæmorrhage.[c] Dr. Lyons recognises but one eruption in typhus which 'may be called indifferently either maculæ or petechiæ,' and yet states that these petechiæ disappear upon pressure.[d] But if we turn to systematic writers on diseases of the skin, we find that petechiæ are defined to be minute purplish spots or sub-cutaneous ecchymoses, which do not disappear upon pressure,[e] and this is now the common acceptation of the term. Although petechiæ, as thus defined, are often developed in the

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[a] Nicholas Massa, 1556. [b] Sennertus, 1619.

[c] 'On appelle généralement du nom de pétéchies, deux affections sympto-
'matiques très distinctes, bien qu'elles aient le réseau muqueux de la peau pour
'siège commun. L'une est une véritable exanthème; l'autre, une hémor-
'rhagie sous-épidermique. Je conserverai le nom de pétéchies à l'exanthème
'et j'appellerai l'hémorrhagie, pourpre, ou taches pourprées. Les pétéchies
'peuvent être considérées, comme le symptôme le plus habituel du typhus.'
Dict. de Méd. 1841. *Art. Pétéchies.* p. 134. [d] Lyons, 1861, p. 121.

[e] 'The term *purpura*,' says Bateman, 'is appropriated by Dr. Willan to an
'efflorescence consisting of small, distinct, purple specks and patches, attended
'with general debility, but not always with fever. The specks and patches
'here mentioned are *petechiæ and vibices*, occasioned, not as in the preceding
'exanthemata, by an increased determination of blood into the cutaneous

centre of typhus-spots, they are not essential or peculiar to typhus. In many cases of typhus, the eruption never becomes petechial, and in few are true petechiæ seen except in the last stages; while, on the other hand, petechiæ are observed in the course of many other diseases, both febrile and non-febrile. Febrile symptoms with petechiæ do not constitute typhus, the peculiarity of which consists in an eruption which often becomes converted into petechiæ. Many of the early writers described the various stages of the typhus-eruption with wonderful accuracy, but the *conversion* of the spots into petechiæ was first noted by Staberoh,[f] Stewart,[g] and and Jenner.[h]

The records of the London Fever Hospital, during a period of ten years, have been examined, to ascertain the frequency of the occurrence of the eruption. Of 3506 cases, the eruption was noted in 3103, and was not observed in 403 cases, or in 11·5 per cent. It was as often absent in males as in females; thus of 1737 males it was absent in 193 (11·11 per cent.), and of 1769 females in 210 (11·87). In children, it was oftener absent than in adults. Thus, the mean age of the patients, in whom the eruption was present, was 29·74, and of those in whom it was absent only 26·28. Again of 398 cases, where there was no eruption, 119 or 30 per cent., were below fifteen years of age, while of 3,058 cases with the eruption, only 444, or 14 per cent., were below fifteen years. In other words, of 563 cases below fifteen years, the eruption was absent in 119, or in 21 per cent., whereas of 2,943 cases above fifteen, it was observed in all but 279 cases, or 9 per cent.; and of 17 cases below five years, it was absent in 7. These figures exaggerate the proportion of cases with no eruption, for in some it had no doubt disappeared before the patient's admission, and in others, where there was only slight mottling, it had probably escaped observation. Of 90 cases noted by myself in 1856, the eruption was present in all but six patients, three of whom did not come under observation until a late period of the disease; and of 1107 cases admitted into the Fever Hospital during the first six months of 1862, the eruption was noted in all but 51. Jacquot observed the eruption in 152 of 159 cases in the Crimea,[i] and Robert Paterson in

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'vessels, but by an extravasation from these vessels under the cuticle.' (*Pract. Synops. of Cut. Dis.* 5th ed. Lond. 1819, p.103, *Atlas*, 1817, Pl. 28). According to Erasmus Wilson, 'When the sanguineous spots (of purpura) are 'minute, they are termed *petechiæ*, but when of large size, *ecchymoses*.' (*Dis. 'of the Skin*, 3rd. ed. 1851, p.337). Dr. Jenner defines a petechia as 'a 'dusky crimson or purple spot, with defined edges, unaffected by pressure, 'and not elevated above the skin.' (JENNER, 1850, xx. 419).

[f] STABEROH, 1838, p.427. [g] STEWART, 1840, p.317. [h] JENNER, 1849.

[i] JACQUOT, 1858, p.172.

110 out of 114 cases at Edinburgh in 1847.[k] Jenner found it in every one of 76 cases above twenty-two years of age, but failed to find it in 13 of 55 cases of the age of fifteen and under.[l]

In children, the spots rarely become petechial; but I have known the eruption perfectly characteristic at every age, from eighteen months to eighty-four years.

Flea-bites have often been mistaken for typhus-spots, but, with a little care, are readily distinguishable by their more circular outline, the minute dark dot in their centre, and by their disappearing on pressure, excepting the central dot.

Fracastorius fixed the first appearance of the eruption at between the fourth and seventh day, a statement which has been endorsed by most subsequent observers. Dr. Stewart analysed 52 cases with this object in view, and ascertained that: 'in more than 'half of the entire number, it appeared on the fifth or sixth 'days, and in exactly three quarters, it appeared from the fourth 'to the seventh day. Taking an average of the whole, it appeared 'most commonly on the sixth day.'[m] Dr. Peacock ascertained the date of the first appearance of the eruption in 28 cases: in 2, it appeared on the second or third day; in 3, on the fourth; in 5, on the fifth; in 7, on the sixth; in 6, on the seventh; in 2, on the eighth; in 2, on the ninth; and in 1, on the ninth or tenth.[n] Of course, those cases only are available for deciding the question, where the eruption first appears while the patient is under observation. According to my experience, the eruption seldom appears later than the fourth or fifth day, and most commonly it is visible on the fourth day. I have rarely met with a case, in which I could be certain, that the eruption made its appearance later than the sixth day. Of 64 cases, in which I specially noted the point in 1856, 37 were admitted into hospital after the sixth day, and the eruption was present in all at the time of admission; in 12, admitted on the sixth day, the eruption was likewise present on admission, and, in 6, it was copious; in 6, admitted on the fifth day, it was present on admission, and in 2 copious; of 3, admitted on the fourth day, in 2, the eruption was present, and, in 1, it appeared on the fifth day; in 3 cases, admitted on the third day, it appeared on the day following, and, in 1 case, admitted on the second day, it appeared on the third day. In many other cases, observed since this calculation was made, the eruption has first appeared on the fourth or fifth

[k] R. Paterson, 1848. [l] Jenner, 1849, xx. 457. [m] Stewart, 1840, p. 318. [n] Peacock, 1856.

day. Cases where it appeared as early as the third day are mentioned by Roupell,[o] Jenner,[p] and W. T. Gairdner.[q]

The average duration of the eruption may be said to be from seven to ten days. In uncomplicated cases, it continues, as a rule, until death or recovery; but sometimes, especially when there is only faint mottling, it begins to fade after a few days, or even hours, and quite disappears a day or two prior to the cessation of the primary fever. On the other hand, when the eruption is dark or petechial, it may linger for a few days after the commencement of convalescence. In cases characterized by both mottling and distinct spots, the former may disappear after a day or two, while the spots continue growing darker until the termination of the case. At Edinburgh, according to Dr. W. T. Gairdner, the eruption has been marked, of late years, by earlier appearance and disappearance than formerly.[r]

The eruption of true typhus never appears in successive crops. Fresh spots may come out for a day or two after their first appearance, but they are superadded to the first spots, and do not take their place. This is the result of my observation in a large number of cases, where I have surrounded every spot with a circle of ink, in order to satisfy myself of the point. Similar observations have been made by Stewart,[s] Jenner,[t] Barrallier,[u] and, indeed, by almost every recent writer on typhus, both English and continental. To quote from Barrallier, (p. 76) 'Toutes apparaissent dans le premier, 'le deuxième, ou troisième jour de leur manifestation; après ce 'temps, il ne s'en montre plus de nouvelles.'

2. *General Hyperæmia* of the Skin. The first appearance of the typhus-eruption is occasionally preceded or accompanied by a pinkish erythematous flush, disappearing on pressure, but returning immediately after. This flush is apparently due to active hyperæmia. In the more advanced stages of severe cases, the surface often exhibits a leaden or livid hue, more especially on the dependent parts of the body. Here there is passive hyperæmia, or stagnation of impure blood in the cutaneous capillaries, resulting from the enfeebled state of the circulation.

3. *Purpura-Spots and Vibices* are sometimes observed in severe cases of typhus, especially when complicated with scurvy. They were particularly common in the Crimea,[x] where typhus and scurvy so often co-existed. These purpura-spots must not be confounded with the petechiæ, already described. Although both

[o] Roupell, 1839, p. 37. [p] Jenner, 1853, p. 285. [q] Gairdner, 1859, p. 51.
[r] Ibid. [s] Stewart, 1840, p. 317. [t] Jenner, 1849. [u] Barrallier, 1861, p. 76.
[x] Jacquot, 1858, p. 178.

are really subcutaneous ecchymoses, the spots of purpura are not formed in the centre of typhus-spots, but are independent.

4. *Sudamina* are occasionally, though rarely, observed in typhus, about the end of the second week. Henderson found them in only 3 of 198 cases.[z] According to Jenner, the appearance of sudamina depends on the age of the patients; he failed to find them in any of 26 patients above forty, but found them in 5 of 17 cases below that age.[a] Two cases of sudamina in typhus in persons above forty-five have come under my observation. When present, they are chiefly observed on the abdomen and in the subclavicular spaces. They may, or may not, be associated with perspiration. In several cases, I have found the fluid, contained in the vesicles, to have an acid reaction; and Barrallier has made a similar observation.[b]

5. *Herpes.* Herpetic eruptions on the lips and other parts of the body, are occasionally observed towards the termination of the disease. Jacquot found them in nearly one-fifth of his patients in the Crimea. I have seen herpes break out on the lips, within the first few days, before the appearance of the typhus-eruption, and then it is apt to mislead as to the nature of the case.

6. *Desquamation.* During convalescence from typhus, the skin is often observed to be rough, and the cuticle separates in the form of minute scales. This desquamation is most marked in cases, where the skin has presented a general erythematous flush. Where sudamina have existed, the scales may be large.

7. *The Temperature* of the skin is always more or less elevated, in the early stages of typhus. Sometimes the skin is hot, burning, and pungent to the feel. The actual temperature may exceed the normal standard, by five or six degrees. I have found it as high as 106° Fahr., but as a rule, it does not exceed 103° or 104°; after the first week or ten days, the temperature usually falls gradually until convalescence, and, in cases of extreme prostration, it may even sink below the healthy standard towards the close of the disease. The elevation of temperature is due to the increased destruction of tissue, and to the diminished evaporation from the surface.

8. *Moisture.* The skin is usually dry from the second or third day, until near the termination of the disease. Convalescence is sometimes ushered in by moderate perspiration, while death is often preceded by copious sweats, giving a macerated appearance to the skin. The secreted fluid has an acid reaction, but in two severe cases I have found it alkaline. In two cases, Barrallier found, that, on evaporation, it left a white efflorescence upon the eyelids and

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[z] Henderson, 1839. [a] Jenner, 1849, No. 2. [b] Barrallier, 1861, p. 218.

nose, consisting of acicular crystals, composed of a free acid, fatty matter, and a large proportion of chlorides.[c]

9. *Odour from Skin* (*Typhus-Odour*). A peculiar repulsive odour is given off from the body of most typhus-patients, after the first week. This smell was noted three centuries ago by Salius Diversus,[d] and has been alluded to by almost every subsequent writer. Lind compared it to the 'odour of rotten straw,' or to 'the disagreeable affecting scent from a person labouring under 'the confluent small-pox.[e] Gerhard spoke of it as 'pungent, ammoniacal and offensive.'[f] Barrallier likens it to the odour of rotten straw, or to that given off by deer, or by certain reptiles, or by rubbing the leaves of rue between the fingers.[g] By other observers, it has been more aptly compared to the smell of mice, but perhaps it is more correct to speak of it, as *sui generis*. It must not be confounded with the smell resulting from the urine being passed in bed, or with the putrid odour which sometimes precedes death from many diseases. The nurses in the Fever Hospital are quite familiar with the typhus-odour, and I have known them distinguish typhus by it alone. The odour is always strongest in damp weather, and when the ventilation is bad. As already stated, there is reason for believing that the typhus-poison is contained in this odoriferous substance.

10. *Altered Cutaneous Sensibility.* (See *Nervous System.*)

11. *Bed-Sores.* (See *Complications.*)

c. Symptoms referrible to the Circulating System.

1. *The Pulse.* As a rule, after the first five or six days, the pulse varies from 100 to 120, and rises with the severity of the general symptoms. It may rise to 150, or upwards; but if it exceed 120 in an adult, the case is severe. Of thirteen cases observed by Henderson, where the pulse exceeded 134, five died, or 38 per cent. Sometimes, on the other hand, the pulse, through the whole course of the disease, never reaches 100, or even 90, and in more than one case, I have known it not to exceed 40 for several days, while the rash persisted, and the tongue was dry and brown; and then it gradually rose to the normal standard, as the other symptoms improved. Barrallier met with this slow pulse, in several cases of typhus; in one case, a man aged fifty-five, the pulse remained for three days at 28.[h] Similar cases were recorded in 1853, by Dr. H. Kennedy.[i] In these cases, the heart's action may be correspondingly slow, or the heart may beat twice

[c] Barrallier, 1861, p. 247. [d] Salius Diversus, 1584. [e] Lind, 1763, p. 62.
[f] Gerhard, 1837, xx. 298 [g] Barrallier, 1861, p. 223. [h] Ibid. p. 70, 87, 248.
[i] H. Kennedy, 1853.

for every stroke of the radial pulse, both conditions indicating that its action is greatly impaired. Of ninety cases of typhus, in which I noted the pulse daily, it never reached 100 in nineteen, rose to between 100 and 120 in seventeen, to 120 in thirty-nine, and to above 120 in fifteen.

Although a rapid pulse is, to some extent, a sign that a case is severe, a slow pulse does not necessarily indicate a mild attack. The cases, where the pulse is remarkably slow, are usually characterized by extreme prostration. Of eleven fatal cases, in which I noted the pulse, it never exceeded 96 in one, it reached 100 in one, 104 in one, 120 in three, 130 in three, and 150 or upwards in two.

The pulse may reach 120 on the third or fourth day of the disease; but usually it does not exceed 100, during the first five or six days. It does not vary, from day to day; but it keeps at the rate which it has once attained, or it continues to increase until death or recovery. A favourable change in the disease is usually marked by a sudden and considerable fall in the pulse. During convalescence, the pulse occasionally falls to below the normal standard, even when it has previously been very rapid. I have repeatedly known it to remain for several days below 50. If the pulse rise much, after falling, it denotes the advent of some complication.

At the commencement, the pulse is full, soft, and compressible, and day by day, as it becomes quicker, it also becomes smaller and weaker, until at last it may be quite imperceptible. In some cases, I have found the pulse to be imperceptible, for several days prior to death. As the pulse diminishes in frequency, it usually increases in volume and force. In young robust persons of sanguine temperament, the pulse during the first week may be firm and somewhat bounding; but in true typhus, this is a rare phenomenon: it occurred only four times in ninety cases, in which I noted its characters, and in three of the four cases there was acute delirium. Most of the cases described in former days by Welsh, Armstrong, etc., as having a pulse of this character, were probably examples of relapsing fever or of acute inflammations.

Now and then, the pulse is observed to be irregular, intermitting, or dicrotous. These characters always point to a very weak condition of the heart. Dr. Lyons has called attention to a very singular want of uniformity, in certain cases, of the force and volume of the arterial pulse in different parts of the system, the carotid, temporal, or iliac arteries, or the abdominal aorta, acting with great violence, while the other arteries are not sensibly disturbed.[k]

[k] Lyons, 1861, p. 155.

Another character of the pulse, observed both during the fever and in convalescence, is its acceleration and diminution in power, on assuming the erect, or semi-erect posture. As Dr. Graves[1] pointed out, the greater the difference, the greater is the debility of the patient.

2. *Action of the Heart.* The state of the heart should be carefully noted in every severe case of typhus, for this organ and the arterial pulse furnish the chief indications for treatment. It is to Dr. Stokes, that the profession is indebted for pointing out the cardiac phenomena of typhus. The principal of these phenomena are a diminution of the impulse, and an impairment, or loss, of the first sound.[m] In mild cases, the impulse and sounds may remain unaltered, but in most severe cases, particularly in persons above thirty, the impulse diminishes progressively from the fifth or sixth day, to the termination of the disease, and for several days prior to death or recovery, it may be entirely absent. At the same time, the systolic sound of the heart, especially over the left ventricle, becomes daily more feeble, and ultimately may be quite inaudible, leaving the second sound clear and distinct. Before the first sound is altogether lost, it may be so short that it is difficult to distinguish it from the second, and then, if the cardiac action be rapid, the sounds may closely resemble those of the fœtus in utero.

The arterial pulse is not an infallible guide to the condition of the heart, which, in all severe cases, should be investigated, by the application of the hand and stethoscope. Although a small, weak, or imperceptible pulse, is usually associated with a diminution of the cardiac impulse and systolic sound, the arterial pulse may be distinct, and not very weak, while the action of the heart is much enfeebled. On the other hand, the cardiac impulse may appear so strong as to distress the patient, and the sounds be distinct, and yet the radial pulse may be imperceptible. Dr. Stokes gives the particulars of a case, where this state of matters lasted for ten days prior to death.[n]

All these abnormal phenomena result from a weakened condition of the central organ of circulation, which depends, as we shall find, on disease of its muscular tissue. They constitute the best and safest guides to a liberal exhibition of stimulants. The state where the cardiac impulse is strong and jarring, but the radial pulse, weak or absent, also demands stimulants; the con-

[1] *Dub. Hosp. Reports*, 1830, v. 469.

[m] For a full account of these phenomena, see STOKES, 1839; GRAVES, 1848, i. 249; HUSS, 1855, p. 74; BELL, 1860; LYONS, 1861, p. 152; also STOKES, *On Diseases of the Heart*, 1854, p. 366.

[n] *Diseases of the Heart*, 1854, p. 384.

tractions of the heart, though violent, are incomplete, and do not suffice to propel the blood, with any force, into the nearest arteries, while, at the same time, there is usually great prostration of the nervous and muscular systems.

3. *Blood.* (See *Post-Mortem Appearances*).

d. Morbid Phenomena of the Respiratory System.

1. *The Respiratory Movements*, in the first week, are usually but little altered, or do not exceed 20 or 24, in the minute; but with the supervention of delirium, and the increased frequency of the pulse, they often rise to 30, or even higher, independently of any pulmonary complication. On the other hand, in cases characterized by great prostration and impairment of the heart's action, especially when the pulse is abnormally slow, the respirations may sink to 8 in the minute.[o]

In other respects, the respiratory act is usually normal; but in grave cases, it may be sighing, irregular, spasmodic or jerking. Spasmodic, or jerking respiration is observed in cases of great cerebral disturbance, and is apt to be followed by coma. There is another variety of the respiratory movement, which is a very unfavourable symptom. This is the 'nervous respiration' of Dr. Corrigan,[p] where the breathing is blowing or hissing, while the mouth is kept closed, the cheeks puff out, and the nostrils dilate with each expiration. The breathing is then often irregular, a long pause being followed by a deep inspiration, and this by a number of other short and rapid inspirations. In some cases of nervous breathing, the action is entirely diaphragmatic, the thoracic muscles being apparently paralysed. All these abnormal characters of respiration may be independent of any pulmonary complication.

2. *The Voice.* As the prostration increases, the voice becomes feeble, and the utterance slow. In the advanced stages, the power of speech may be entirely lost.

3. *Cough and Expectoration.* (See *Pulmonary Complications*).

4. *The Expired Air* in typhus is remarkable for its disagreeable odour, which is most marked in the advanced stage, and in severe cases. It has been compared to yeast, but it often closely resembles the peculiar odour exhaled by the skin (see page 134). More careful examination has shown, that this air differs from that expired in health, in containing a smaller quantity of carbonic acid, and a larger amount of ammonia.

Twenty years ago, Dr. A. G. Malcolm, of Belfast, recorded the

[o] See Dr. John Reid's *Anat. and Path. Res.*, p. 206. [p] Corrigan, 1853, p. 72.

results of upwards of fifty experiments on the air expired by patients labouring under typhus, with the object of ascertaining the amount of carbonic acid.[q] These experiments were performed with Dr. Prout's apparatus, and seem to have been done with great care. The results were very uniform, and were compared with those obtained by Dr. Prout, from examining the air expired by persons in health. According to Prout, the proportion of carbonic acid is 3·96 per cent. of the whole air exhaled in health. This is probably a low estimate. In some of the experiments of Messrs. Allen and Pepys, it was as much as 8 per cent.; and about 4·35 per cent. may be taken as the average of of the results obtained by different observers.[r] But in typhus, Dr. Malcolm found that the quantity was invariably reduced; in one case it did not exceed 1·18 per cent., while the average of forty-five examinations was only 2·492 per cent. He also ascertained, that the quantity was smallest in the more severe forms of the disease, characterized by delirium, subsultus, and dry brown tongue. Vierordt has shown that, even in health, the proportion of carbonic acid in the expired air diminishes as the frequency of the respirations increases. This observation may partly account for Dr. Malcolm's results in typhus, but not entirely, for, even when Vierordt breathed ninety-six times in the minute, the quantity only fell from 5·5 to 2·6 per cent., while in one of Malcolm's cases, the respirations were 24, and the proportion of carbonic acid only 1·77. But, accepting this explanation, the fact remains, that in typhus the quantity of carbonic acid, eliminated from the lungs, is diminished.

In 1854, Professors Viale and Latini of Rome,[s] after a series of very careful and delicate experiments, confirmed the statements of Marchand and Reade,[t] to the effect that small quantities of ammonia are constantly evolved with the expired air in health, and showed that, in some contagious diseases, more especially typhus, this quantity was much increased, while, at the same time, ammonia was given off by the skin, and with the other excretions. From their observations they inferred, that the active principle of contagion was probably some ammoniacal salt. In the same year, Dr. Reuling pointed out, that the air expired in certain diseases, such as typhus, uræmia, and pyæmia, contained an excess of ammonia.[u] These results have since been confirmed, by the independent and elaborate researches of Dr. Richardson. It

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[q] Malcolm, 1843. [r] Carpenter's *Princ. of Hum. Phys.*, 5th ed. p. 283.
[s] Viale and Latini, 1854. [t] See page 115.
[u] Lehmann's *Phys. Chem.* (Day's *Trans.*), iii. 559.

is well known that, in severe cases of typhus, the breath has often an ammoniacal odour, and that thick white fumes are produced, on holding a glass rod, previously dipped in hydrochloric acid, close to the mouth of the patient. Dr. Richardson found the breath in one case so ammoniacal, that it coated a glass slide, moistened with hydrochloric acid, with crystals of chloride of ammonium, and restored the blue colour to reddened litmus paper.[x] I have examined the breath in a large number of cases of typhus, and in grave cases, with typhoid or putrid symptoms well developed, I have rarely failed to obtain the crystals of chloride of ammonium, in the manner indicated. Sometimes, not more than a dozen expirations on the glass slide sufficed to produce a large number. They were mostly dendritic, as represented in the annexed woodcut. Similar crystals may be obtained, by adding hydrochloric acid to a solution of carbonate of ammonia.

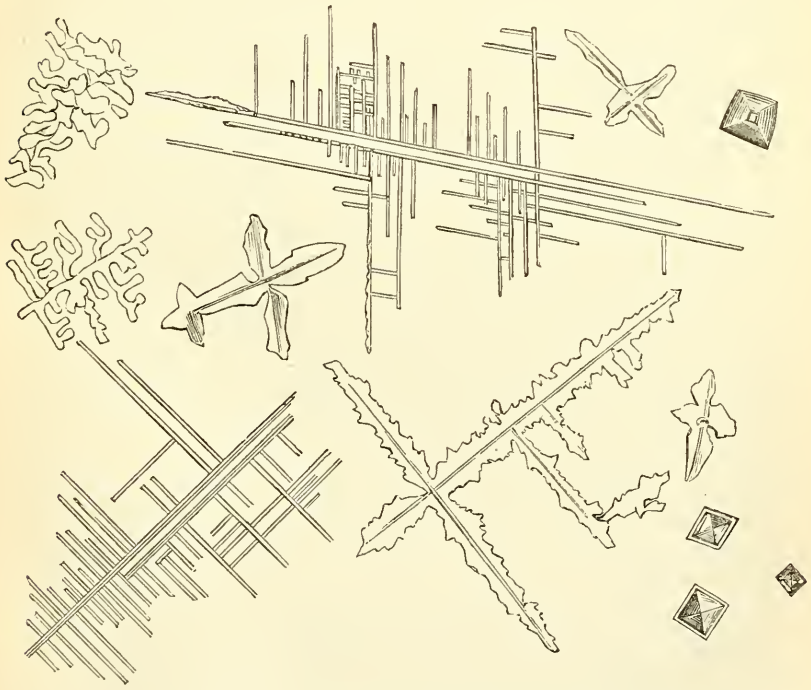

Fig. 2. Crystals of Chloride of Ammonium obtained from the breath of a patient suffering from Typhus. The octohedral crystals to the right were probably chloride of sodium, derived from the acid.

[x] Richardson, 1858, p. 346.

e. Morbid Phenomena presented by the Digestive Organs.

1. *The Tongue* is at first covered with a creamy white fur, which gradually increases in thickness, and after a few days often assumes a dirty-yellowish aspect. At the same time, the impressions of the teeth, along the margin, show that the organ is enlarged. In mild cases, the tongue may remain moist and furred, throughout; but usually, about the end of the first week, it becomes dry, rough, and brownish, along the centre. Still later, in severe cases, it contracts into a ball, and is covered with a dry, dark-brown, or black, cracked crust. Of 90 cases, in which I have noted the appearances presented by the tongue, it became dry, dark-brown, or black all over, at some period of the disease, in 53; in 14 cases, it was merely dry, rough, and brownish along the centre; and in 23, it was covered with a thick, white or yellowish, moist fur, through the entire course of the disease.

The amount of dryness and darkness of the tongue is a fair criterion of the severity of the case. Of the 90 cases above referred to, all that were fatal (11), except one, had the tongue dry and dark, and in the exceptional case death was due, not to the primary fever, but to complications during convalescence: the cases where the tongue never became dry were all of a mild character. With the first commencement of convalescence, the dry brown tongue becomes clean and moist at the edge, and the brown crust is gradually removed.

The colour of the tongue at the margin and tip is usually pale; but occasionally it is red, and the papillæ are enlarged. The crust, which covers the hard brown tongue, is often irregularly cracked, but the tongue itself is not often fissured. The deep transverse fissures, so common in pythogenic fever, are rare in typhus. Still more rarely is the tongue in typhus red, smooth, glazed and fissured. In 90 cases, I found the tongue fissured only twice; and in 41 fatal cases, Dr. Jenner found it fissured only four times.

In many cases, the tongue is tremulous; while in some, it is retracted and cannot be protruded. These phenomena usually co-exist with the dry brown tongue, but may be independent.

2. *Brown Sordes* usually begin to collect upon the teeth and lips, about the beginning of the second week in severe cases. These sordes, as well as the brown crust on the tongue, consist of an accumulation of epithelial debris, which becomes black from

desiccation, or sometimes from admixture of blood. In rare cases, hæmorrhage from the gums has been observed.[y]

3. *Loss of Appetite* is one of the earliest and most constant symptoms of typhus, and lasts until the disease takes a favourable turn. Occasionally, a demand for food is the first and only symptom of returning health; and in most cases, such a demand is to be regarded as a favourable indication, although no improvement has taken place in the other symptoms.

4. *Thirst* is present to a greater or less degree in all cases. In 22 out of 90 cases, I have noted it as excessive. It is usually greatest during the first week, and, in the stage of nervous prostration, it abates or ceases.

5. *Dysphagia.* See *Muscular Paralysis* p. 159, and *Pharyngitis* under *Complications*.

6. *Nausea and Sickness* are not common symptoms. Vomiting occurred in 9 out of 90 cases, of which I have notes; in none, was it severe. The vomited matters consisted, for the most part, of a green bilious fluid. In 4 of the cases, slight vomiting was one of the primary symptoms, and did not recur after the second or third day. In 2 other cases, it was likewise a primary symptom, and ceased on the second day, but recurred at intervals, from about the twelfth day until convalescence. In the 3 remaining cases, it was only observed in convalescence, and was due to other causes than the primary fever.

Of 198 cases of typhus observed by Henderson in the Edinburgh Infirmary, in 1838-9, nausea and vomiting occurred in only 12, chiefly at the beginning of the fever.

7. *Meteorism* is also a rare symptom in typhus. In 5 only out of 90 cases, have I noted the abdomen as abnormally tympanitic or distended, while in many it was flat or even concave. The late Dr. Todd[z] believed that meteorism was more common in typhus than in pythogenic fever, and a similar opinion is expressed by Dr. Austin Flint;[a] but their view is contrary to the experience of most observers. In 3 only out of 41 fatal cases was the abdomen observed by Jenner, to be unnaturally distended. Marked tympanitis was observed by West,[b] in 11 out of 60 cases; by Henderson,[c] in 8 out of 198; by Stewart,[d] in 15 out of 139; by Shattuck,[e] in 1 of 9; and by Barrallier,[f] in 4 of 1312. Adding these results to those obtained by myself, we have 1849 cases, of which meteorism occurred in 47, or in 1 of 39·34. Excluding

[y] Barrallier, 1861, pp. 236, 360. [z] Todd, 1860, p. 168. [a] Flint, 1852.
[b] West, 1838. [c] Henderson, 1839. [d] Stewart, 1840, p. 310.
[e] Bartlett, 1856, p. 199. [f] Barrallier, 1861, pp. 239, 361.

M. Barrallier's cases, which may be thought to have an undue preponderance, there remain 534 cases, of which meteorism was observed in 43, or in 1 of 12·4. In the Crimean typhus, meteorism was observed by Garreau, in 1 out of 8 cases, and by Mouchet, not at all: Jacquot noted it in about one-third of his cases.[g]

In the few cases, where meteorism is met with, it occurs at an advanced stage; it is associated, not with abdominal pain or diarrhœa, but with great prostration and cerebral oppression; and then, like the meteorism, so common in paraplegia, it depends on want of nervous tone in the coats of the bowel, and is always a bad symptom. In rare cases it is excessive, so as even to interfere with respiration.

8. *Gurgling*, on pressure of the abdomen, is quite exceptional. In the few cases where there is diarrhœa it may be detected; but then it is not usually confined to any part of the abdomen. Of 43 fatal cases, Dr. Jenner discovered gurgling in only one.

9. *Abdominal Pain and Tenderness* are rarely complained of. Occasionally, during the first week, there are colicky pains, which are transient, and not attended by tenderness on pressure. Vomiting is sometimes associated with pain and tenderness at the epigastrium; and a distended bladder, with pain and tenderness in the hypogastrium. But abdominal tenderness in typhus is never limited to, or most marked in, the cœcal region, nor is it accompanied by diarrhœa or tympanitis.

10. *The Liver and Spleen* are in some cases found to exceed their normal boundaries on percussion. In 1202 cases observed by Barrallier, the liver was slightly enlarged in 365, or 30·3 per cent.; and the spleen, in 126, or 10·48 per cent.[h] It is rare that either organ is the seat of pain or tenderness.

11. *Constipation* is the rule in typhus; and diarrhœa, the exception. Of 144 cases in which I have noted this point, the bowels were constipated, so as to require the administration of laxatives, in 78. They were opened once (33) or twice (18) a day in 51, and, in many of these cases, purgatives had been taken before admission into hospital; in connection with which it should be observed, that in several cases of constipation, where laxatives were taken after admission, the bowels continued open afterwards. In only 15 cases, was there any approach to diarrhœa; and only in 6, was it necessary to have recourse to astringents. In 4 of the 15 cases, diarrhœa was one of the earliest symptoms, and was associated with sickness, but both symptoms may have resulted from medicine; in a fifth case, vomiting had been one of the

[g] Jacquot, 1858, p. 185. [h] Barrallier, 1861, pp. 240, 361.

primary symptoms, and the diarrhœa came on about the tenth day, after a purgative; in 9 cases, there was diarrhœa, for a day or two, at the period of crisis, about the end of the second week, the bowels having previously been constipated; in the remaining case, the bowels were confined throughout the primary fever, but diarrhœa and sickness supervened in convalescence, during the separation of a large slough over the sacrum. Of 43 fatal cases observed by Jenner, spontaneous diarrhœa occurred in only 4.[i] Of 154 cases, noted by Henderson at Edinburgh, the bowels were costive in 50, easy in 99, and loose in 5.[k] Of 139 cases observed by Stewart at Glasgow, there was costiveness (notwithstanding purgatives) in 62, a relaxed condition after medicine in 53, while only in 24 were the bowels spontaneously relaxed[l] Of 1302 cases, observed at Toulon by Barrallier, there was constipation in 769, diarrhœa in 136, and regularity of the bowels in 397.[m] Adding these results to my own, it appears, that of 1782 cases, diarrhœa occurred in only 184, or in 10·32 per cent., while in 959 of 1739 cases, or 55·14 per cent, there was obstinate constipation. It may be added, that the occurrence of diarrhœa in typhus, in the French army in the Crimea, was quite exceptional.[n] Lastly, the practice of treating typhus by purgatives, recommended by the late Dr. Hamilton, of Edinburgh,[o] may be cited as another proof, that this fever is not often characterized by diarrhœa.

Spontaneous diarrhœa in typhus is chiefly observed, as an occasional symptom about the period of crisis. It may then, in rare cases, be excessive, and cause such an increase of prostration, as to endanger for the first time the life of the patient; but it is never, as far as my experience goes, attended by abdominal pain or tenderness, whatever be the stage at which it occurs. It should be remembered, that, in the advanced stage of typhus, the frequent passage of liquid stools may be due to paralysis of the intestines and sphincter, and to the fluid character of the ingesta.

12. *Characters of the Stools.* When there is no diarrhœa, and when the bowels are moved spontaneously without medicine, as happens in 34½ per cent. of cases (see above), the stools are usually of normal consistence and colour, but sometimes they are darker than natural. When there is diarrhœa, either spontaneous, or from medicine, they are mostly of a dark-greenish brown colour, and not very watery. The reaction of the stools is usually acid, as in health;

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[i] JENNER, 1849. [k] HENDERSON, 1839. [l] STEWART, 1840, p. 308.
[m] BARRALLIER, 1861. pp. 240, 361. [n] JACQUOT, 1858, pp. 185, 201.
[o] HAMILTON, 1805.

but the (spontaneous) relaxed stools, which are most common at an advanced stage, were found to be strongly alkaline, in two instances, by Dr. Parkes, probably owing to the presence of ammonia. In many cases, the relaxed stools contain numerous crystals of ammoniaco-magnesian phosphate.[p]

13. *Intestinal hæmorrhage* is extremely rare. Dr. Jenner states, that of nearly 2,000 cases of typhus, of which notes were taken at the London Fever Hospital during three years, the only instance, in which bleeding from the bowels occurred, was that of an old man who had hæmorrhoids. Out of upwards of 1000 cases of typhus which have come under my own care, I have never met with an example of intestinal hæmorrhage.

But though intestinal hæmorrhage is rare in typhus, its occurrence is not impossible. Dr. Tweedie mentions a case of well-marked typhus, where hæmorrhage from the bowels was the apparent cause of death. Peyer's patches and the solitary glands were healthy, and there was no enlargement of the mesenteric glands; but the mucous membrane of the ileum, and the commencement of the colon was red and tumid.[q] Frerichs records a case of 'typhus exanthematicus' complicated with jaundice, where extensive hæmorrhage from the bowel occurred, followed by great exhaustion. There were a few 'hæmorrhagic erosions' found after death in the rectum, but the ileum and mesenteric glands were healthy.[r] Barrallier observed extensive hæmorrhage from the bowels, in two of 1058 cases of typhus.[s]

The circumstance that, in some epidemics, typhus is complicated with scurvy or dysentery, coupled with the non-recognition of the distinction between typhus and pythogenic fever, may account for the frequency, with which intestinal hæmorrhage has been observed by some Irish physicians. Dr. H. Kennedy,[t] however, states that he has met with 30 cases of intestinal hæmorrhage in typhus, and that no ulceration of the bowel was found in those which were fatal.

f. Morbid Phenomena presented by the Urinary System.

1. *The urine* undergoes important changes in typhus.

The quantity varies, to a certain extent, with the amount of fluid ingesta, but during the first week, it is sometimes diminished by one-fourth or one-half, notwithstanding the dryness of the skin and the large amount of fluids drunk. There appears to be

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[p] PARKES, 1850, p. 396. [q] TWEEDIE, 1860.
[r] FRERICHS, *Dis. of Liver*. Syd. Soc. Tr. i. 168. [s] BARRALLIER, 1861.
[t] H. KENNEDY, 1860.

an absolute retention of water in the system. In the advanced stage, there is occasionally complete suppression of urine; but this is fortunately rare, as it is almost always fatal. More commonly, the quantity increases in the later stages. I have repeatedly found that a large quantity of pale, limpid urine, of low specific gravity, was passed during the 'typhoid stage.' With the commencement of convalescence, the quantity is greatly increased.

The Colour is usually dark, often reddish-brown, in the early part of the disease, but afterwards it becomes paler. At the commencement of convalescence, as the quantity increases, the urine becomes pale-yellow and limpid.

The acidity is marked, in the early stage; but in the second week, it becomes more feeble, while sometimes the urine is neutral or alkaline, and deposits phosphates. This alkalinity, however, is usually due to decomposition, either in the bladder, or shortly after micturition.

The specific gravity varies with the amount of water, and with the stage of the disease. In the early stage, it is usually high (1024-30); but, as the disease advances, it gradually falls. With convalescence, there is often a sudden fall; the density may then, for several days, not exceed 1003.

The total daily amount of urea, excreted in the urine, is increased.[u] In 1857, Dr. Parkes[x] published the results of a daily analysis of the urine, in a case of typhus, commencing with the eighth day, and ending with the twenty-sixth. The daily amount of urea during the fever was increased by one-fifth, or averaged 530 grains, the healthy average being only about 400 grains. On one day, when 120 grains of extract of coffee were given, the quantity rose to 723 grains. Otherwise, the amount was remarkably regular from day to day; and it continued large for some days after the temperature had fallen to below the normal limits. It fell during convalescence to 336 grains, but in two or three days rose again to the healthy standard. My colleague, Dr. Buchanan, has lately made observations on the daily amount of urea excreted, in a considerable number of cases of typhus. The results are not yet published, but the quantity was always much increased.

[u] Haller states, that the quantity is diminished, but I have seen no account of his experiments (HALLER, 1853). M. Barrallier concludes, from observations on the urine of prisoners suffering from typhus, in the hulks at Toulon, that there is a progressive diminution of urea from the earliest stage (BARRALLIER, 1861, pp. 141, 251 and 366). Barrallier, however, does not seem to have ascertained the absolute amount of urea, but only the proportion in 1000 parts of urine.

[x] PARKES, 1857; and *On the Urine*, 1860, p. 258.

From a few isolated observations of my own, I am inclined to think that, as in enteric fever, the increase of urea is greatest in the early stage, and that, after the eighth day, it progressively diminishes. It is important to note, that on the commencement of convalescence, in Parkes's case, there was a sudden fall in the amount of urea, which, for several days, was below the normal standard.

As in other febrile conditions, the increased formation of urea, notwithstanding the diminished supply of food, is evidently the result of an exaggerated disintegration of the muscular and other nitrogenous tissues. As long as the urea continues to be eliminated by the kidneys, its effects are comparatively trifling; but if the quantity be excessive, and still more, if, from any morbid condition of the kidneys, either antecedent to, or resulting from, the febrile attack, its elimination be interfered with, it accumulates in the blood, and gives rise to uræmic (typhoid) symptoms. If the urine be completely suppressed, as sometimes happens, death speedily ensues, under symptoms of coma, and sometimes with uræmic convulsions; but if the elimination be less complete, it may still give rise to delirium, stupor, and coma. Indeed, it is very possible, that these symptoms, so characteristic of typhus, are in a great measure due to the presence of urea, or of its derivative, carbonate of ammonia, in the blood. (See page 12). This supposition is confirmed by the ammoniacal nature of the exhalations. Moreover, urea has been repeatedly found in the blood of persons dying of typhus, with marked cerebral symptoms, even although there has been no disease of the kidneys, and no diminution in the amount of urine. This was proved to be the case in 1844, by Mr. Michael Taylor. A man, aged fifty-three, died on the twelfth day of an attack of typhus; the eruption was well marked. Death had been preceded, for four days, by stupor and muttering delirium. Some hours before death, three pints of urine were drawn off by catheter. After death, the kidneys were found perfectly healthy—not even congested—and urea was discovered, in considerable quantity, in the blood removed from the heart and large veins.[y] Dr. Christison records a case of typhus, which proved fatal on the tenth day, from sudden coma and convulsions, and where urea was found in large quantity in the serum of the blood, the kidneys, with the exception of congestion, being healthy;[z] and Frerichs records cases of both typhus and pythogenic fever, in which death occurred from uræmia.[a] In several cases of typhus, where death has been preceded by profound stupor, I have obtained urea, in considerable quantity, from the serum of the blood. The observations, which

[y] Taylor, 1844. [z] Christison, *On Granular Degen. of Kidneys*, p. 167.
[a] Frerichs, *Die Brightsche Nierenkrank.* p. 210.

have been made in relapsing and pythogenic fevers, also support the opinion, that the head-symptoms in typhus are due, not to inflammation, as was once believed, nor to the presence of the original fever-poison in the blood, but to the circulation through the brain of urea, carbonate of ammonia, or other products of disintegrated tissue.

The uric acid is also increased. Crystals of uric acid are often deposited spontaneously; and, as a rule, are thrown down in large quantity, on the addition of nitric acid. Salts of uric acid, in the form of lateritious sediment, occur at any stage of the disease; they are not necessarily critical; but I have observed them mostly within the first four or five days, or towards the termination of the disease, particularly in cases where the typhoid state has been well marked.

The chlorides gradually diminish from the first, and by the eighth day they are reduced to a mere trace, or may be entirely absent. This diminution is not altogether due to the reduced quantity of salt in the food, for my colleague, Dr. Buchanan, has ascertained that, after administering as much as an ounce and a half of salt by the mouth, about the eighth day of the fever, scarcely a trace of chlorides could be discovered in the urine for several days. (See also Case II. p. 122). The patients, on whom these results were obtained, had no pulmonary complication and no diarrhœa. It would seem, that either the power of absorbing chlorides is impaired, or that, as in the case of pneumonia, there is an absolute retention of them in the system. Whatever be the explanation, the absence of chlorides from the urine is not pathognomonic of pneumonia, as has been imagined.[b]

Albumen is not uncommon in the urine of typhus. Dr. G. W. Edwards came to the conclusion, that the urine almost always becomes albuminous, *at an early period.* Of 14 cases, in which he tested the urine, between the sixth and eighteenth days, albumen was present in all; of 2 cases, examined on the sixth day, there was albumen in 1; and of 6 cases, examined on the seventh day, it was present in all. One of the cases died, and in the remaining 13, the albumen disappeared between the fourteenth and eighteenth days. In 6 other cases, where the urine was tested after the twentieth day, no albumen was found. The quantity of the albumen was in some cases abundant, especially at its first appearance, and as long as the albumen was

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[b] See for example, BENNETT, *Princip. and Pract. of Med.* 2nd ed. p. 638.

present, the specific gravity was usually low.[c] Dr. Sidey found albumen 'in a very large proportion of cases of typhus' at Edinburgh; it occurred invariably towards the crisis of the fever, and, in many cases, on the sixteenth day of the disease.[d] Oppolzer noticed albumen in most cases of exanthematic typhus, and sometimes tube-casts also; the amount of albumen was often as great as in Bright's Disease.[e] Austin Flint found albumen in 7 out of 9 cases in America; in 3, it was present on the first or second day: of the others, where it was not looked for until later, it was found on the sixth day, in 2; on the tenth, in 1; and on the fourteenth, in 1.[f] Moering, a Russian physician, found the urine very often albuminous in the typhus of the Crimea;[g] and Barrallier found small quantities of albumen, in the advanced stages of typhus, at Toulon.[h] Cases of typhus, with albumen in the urine as early as the eighth day, are recorded by Drs. Johnson[i] and Gull.[k] On the other hand, Wunderlich discovered albumen in only 4 out of 49 cases of exanthematic typhus.[l]

During the spring of the present year, I tested the urine daily for albumen in 28 cases, of typhus, from about the sixth to the twentieth day of the disease. The cases were not selected; but the nurse was told, to keep the urine of every patient admitted into hospital with a distinct typhus-rash. In 8 of the cases, no trace of albumen was ever present. All of these cases were mild; none of them presented the typhoid stage well-marked, and all recovered. In 20 of the cases, or 71·4 per cent., albumen was present in greater or less quantity; and of this number, 5, or 52 per cent., died. In 11 of the 20 cases, the quantity of albumen was very slight, and in most cases it was transient, lasting only for a day or two, about the termination of the disease; one of these patients died on the ninth day of the attack; the rest recovered. In 9 cases, the albumen was in considerable quantity, and lasted for several days; in some of the cases, it appeared as early as the seventh day, and lasted until death or recovery. All of these cases were severe; in all, the typhoid state was well-marked, and 4 of the 9 cases, or 44·4 per cent. died. In most, if not all, the cases here analysed, the albuminuria was obviously induced by the febrile attack, and was not the result of any previous renal disease, for it was ascertained to commence during the attack, and in the

[c] EDWARDS, 1853. [d] *Br. and For. Med. Chir. Rev.*, July, 1853, p. 59.
[e] SCHMIDT'S *Jahrbuch*, 1857, No. 11, p. 256. [f] FLINT, 1852, p. 334.
[g] JACQUOT, 1858, p. 203. [h] BARRALLIER, 1861, pp. 251, 367. [i] JOHNSON, 1862.
[k] GULL, *Med. T. and G.*, Ap. 5th, 1862. [l] PARKES *On the Urine*, 1860, p. 260.

cases that recovered, it ceased with convalescence. It would therefore appear, that, in severe cases of typhus, the urine, as a rule, contains albumen, and that when the albumen appears early in the disease, or the quantity is large, the danger to the patient is correspondingly great.

Although albuminuria in typhus is occasionally the result of previous disease of the kidneys, it is oftener due to simple hyperæmia, and to the liquefied condition of the blood, which characterizes typhus, or to actual disease of the renal tissue, induced by the febrile attack. I have often discovered epithelial casts, or sometimes even blood-casts, in the urine, along with albumen. In several instances, also, where death has occurred during an attack of typhus, I have found the kidneys present all the characters of acute nephritis (as in scarlatina), while in many others, where there had been no previous history of renal disease, but where death was due to complications during convalescence from typhus, I have found the kidneys much enlarged (in one case each kidney weighed 8 ounces), smooth and pale, with the capsule non-adherent, the cortex hypertrophied, and the tubes gorged with epithelium. Dr. Johnson mentions two cases, where the kidneys became diseased during convalescence from typhus, one of which proved fatal.[m]

But, whether the albuminuria in typhus result from simple hyperæmia, or from more serious disease of the kidneys, antecedent to, or consequent on, the attack of typhus, it shows, that there is an obstruction to the channel, by which the excessive amount of urea, and other products of retrograde metamorphoses are to be eliminated from the system; and accordingly, the danger increases with the extent and duration of the obstruction, as indicated by the quantity and date of appearance of albumen in the urine. The occurrence of blood in the urine is a still more dangerous sign.

Epithelium. In most cases, the urine throws down a mucous cloud, containing a quantity of vesical epithelium, sometimes mixed with renal epithelium and casts of the uriniferous tubes.

Leucine ($C_{12}H_{13}NO_4$) and *Tyrosine* ($C_{18}H_{11}NO_6$), two products of the disintegration of albumen or fibrine, of a more complex character than urea, have been detected in the urine of typhus by Frerichs, and, in one instance, by myself. (See *Jaundice*, under *Complications*). In most of the cases where they have been found, there has been some morbid change of the liver, and the urine has likewise contained bile-pigment and the bile-acids.[n]

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[m] *Diseases of the Kidney*, 1852, p. 74.

[n] Frerichs, *Diseases of the Liver*, Syd. Soc. Transl. i. 168, 205, and Parkes *On the Urine*, 1860, p. 191.

Sugar has occasionally been found in small quantities in the urine of typhus, by Dr. Buchanan.

2 *Retention and Incontinence of Urine.* (See *Symptoms* under *Nervous System.*)

g. Morbid Phenomena presented by the Nervous and Muscular Systems.

1. *Head-ache* is one of the first and most constant symptoms of typhus. Of 92 cases, noted by myself in 1856, head-ache was complained of in all but 6. Henderson found it in 150 out of 159 cases, at Edinburgh: in 92 out of 108 cases, it was present on the first day; its mean duration was ten days.[o] Stewart noted head-ache after the fifth day, in 98 out of 139 cases at Glasgow; this number was exclusive of the cases, in which the head-ache had ceased before the fifth day.[p]

The head-ache is always most severe during the first week; it often lasts only a few days, and usually it ceases, or greatly abates, with the advent of delirium about the eighth day. In a few cases (1 in 12·5, Stewart), mostly those in which there is no delirium, it is continued through the whole course of the disease. After the complete cessation of the head-ache, the patient may continue to complain of pains in other parts of the body.

The seat of the pain is most often in the forehead or temples; at other times it is general; it is rarely confined to the vertex or occiput.

The severity of the pain varies. In some patients, especially the young and plethoric, it is intense, and for a few days, it is the most prominent feature of the malady; in others, it is comparatively slight.

The character of the pain is usually dull and heavy. The patient is often unable to define it. It is rarely described as darting, stabbing, throbbing, or bursting.

2. *Vertigo.*—The head-ache is, in most cases, accompanied by more or less giddiness, which is aggravated by sitting up, and increases with the progress of the disease.

3. *Pains in the Back and Limbs* are usually present from the first. As a rule, they cease about the end of the first week, but they are often complained of after the cessation of head-ache, and they may recur with some severity during convalescence. The pain in the back is of a dull, heavy character, and rarely approaches in severity to that which precedes the eruption of small pox. The pains

[o] Henderson, 1839. [p] Stewart, 1840, p. 306.

in the limbs resemble those resulting from bruises, or sometimes they are likened to cramps; they are usually more severe than the pains in the back.

4. *Impairment of the Mental Faculties.—Delirium.* The mental faculties are almost invariably more or less affected, in the course of typhus. It is only in exceptional cases of a mild nature, that there is not some mental confusion, while in the majority there is actual delirium. Hence it is, that typhus is often denominated 'brain fever' (see *Synonyms*, p. 18). The frequency and character of these symptoms, however, vary at different times and places, and are much influenced by the previous habits and condition of the patients. When typhus attacks persons in the upper classes of society, or the intemperate, or the subjects of mental anxiety and fatigue, the delirium and impairment of the mental faculties are more constant, earlier in their development, and more marked. Of 90 cases noted by myself at the London Fever Hospital, the mental faculties were impaired in 78, or in 86·6 per cent, while in 52, or 57·7 per cent, there was delirium. Of 198 cases observed by Henderson at Edinburgh, in 1838-9, there was delirium in 48, and in most of the others, there was confusion or sluggishness of mind.[q]

The severity of an uncomplicated case may be measured, by the degree of mental aberration and delirium. Of the 11 fatal cases (included in the above 90), I noted great delirium in 10; in the remaining case, the primary fever was comparatively mild, and death resulted from complications during convalescence. Of 43 fatal cases recorded by Jenner, delirium (28 cases), or mental confusion only (14 cases), was present in all but one patient, who survived the primary fever, and died of secondary phlebitis.[r]

In most cases, it is towards the end of the first week, that the mental faculties become blunted and confused; the patient hesitates and looks stupid when spoken to; he can give no account of his illness; he forgets how long he has been in hospital: or he is even ignorant of where he is, while he is indifferent to all that is passing around, and does not like to be disturbed. At the same time, there is often much moaning and restlessness, with talking in the sleep. In mild cases, this state of mental obfuscation may never be exceeded, but more commonly it is followed by delirium.

Delirium does not usually come on until the end of the first, or the beginning of the second week. Sometimes it does not commence so early: it may supervene at any time during the second

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q HENDERSON, 1839. r JENNER, 1849. No. 2.

week, or it may precede the crisis merely by a day or two. On the other hand, it may commence much earlier. I have had two patients under my care, where the patient was seized with active delirium, on the first night of the attack; in one, the case was at first mistaken for mania. In my own first attack, delirium set in on the morning of the second day, and lasted for twelve days. Jacquot[s] and Barrallier[t] both mention cases, where delirium came on during the first night. Of 1005 patients observed by Barrallier at Toulon, the delirium appeared during the first week in 371, during the second in 602, and during the third in 32.

At first, the delirium shows itself at intervals during the night, or it lasts all night, and by the morning it may have ceased entirely, again to return on the following evening and last through the night. It is surprising how rational persons may be during the day, who in the night are very delirious. By and bye, the delirium becomes more continued, but, as a rule, it is worse at night; or what is very commonly the case, the patients are wakeful and delirious at night, stupid and drowsy in the day-time. After the delirium has commenced, it continues more or less, until death or convalescence, provided it be not succeeded by great stupor or coma. With convalescence, it ceases; but in several cases, I have known it persist for several days, after the pulse had fallen to the normal standard, and there was a general improvement in the other symptoms.

There is no relation between the headache and delirium. In most cases, the former has ceased before the commencement of the latter, a feature of no small importance, as regards diagnosis from cerebral inflammation.

The character of the delirium varies greatly. Most commonly it is of a low form—the '*typhomania*' of Galen and early writers.[x] The patient lies quietly, muttering incoherently from time to time, but he is at first easily roused so as to give coherent answers; or he is restless, irritable, and sleepless, and answers in a rambling, incoherent manner; ultimately, in either case, he becomes torpid, and more or less unconscious. A second form of delirium is of a busy character, and more or less approaches the '*delirium tremens*' of the drunkard. The patient is extremely prostrate, but at the same time restless and fidgety; he sleeps badly, or not at all; he moves about in bed, or he tries to get up, with apparently no

[s] JACQUOT, 1858, p. 164. [t] BARRALLIER, 1861, pp. 231, 360.

[x] The definition of typhomania, given by many of the early writers, is: 'affectus ex phrenitide et lethargo mixtus.' Forestus defined it as 'genus 'delirii cum levi furore mixtum.' (FORESTUS, 1591, ed. 1653, p. 239).

definite object; the pulse is quick and feeble; the cardiac impulse is weak, the skin is moist, and there are tremors of the limbs and tongue. Or, thirdly, the delirium is of an acute and noisy character, the '*delirium ferox*' of some writers. The patient does not sleep; but rolls his head from side to side, shouts and screams incessantly, and makes constant attempts to leave his bed and roam about. His muscular power is often surprising; he will lift heavy weights, and it may require several strong attendants to keep him in bed. At the same time, the pulse is rapid, full, and sometimes of good strength; the cardiac action is violent; the skin, hot and dry; the face, flushed; the conjunctivæ, injected; the eyes, intolerant of light, and the ears, of noise; the physiognomy, bold and excited. In this state, patients often exhibit a suicidal tendency. Very often they attempt to throw themselves from a window, and fatal consequences occasionally result from their succeeding.[y] Barrallier mentions the case of a patient, who inflicted a deep gash in the hypogastric region, in endeavouring to amputate the penis.[z] Bell alludes to a patient, who, fancying that a robber was up the chimney, rose and attempted to climb up, but fell covered with soot, and with his forehead cut against the fire-irons.[a] Among the French troops in the Crimea, it was not uncommon to see patients, in this state, running delirious over the fields;[b] and hence, we can understand the statement, in the account of the Oxford 'Black Assize,' that: 'Some leaving their beds, occasioned by the rage of 'their disease and pain, would beat their keepers or nurses, and 'drive them from their presence; others, like madmen, would run 'about the streets, markets, lanes, and other places; and some 'again would leap headlong into deep waters.'[c] This acute form of delirium is very apt to be followed by profound prostration, or fatal collapse; at other times, it gradually passes into the first form, or typhomania. On the other hand, typhomania, after lasting for several days is, in rare cases, succeeded by 'delirium ferox.'

Every possible gradation between these typical forms of delirium may be encountered. The acute, noisy delirium, however, is comparatively rare. In the Philadelphia epidemic of 1836, according to Gerhard, the delirium was only acute and noisy in one patient out of 20.[d] Of 43 fatal cases, observed by Jenner, only 7 (or 16 per cent.) attempted to leave their beds and roam in the wards.[e] Of 90 cases noted by myself, delirium occurred in 52, but only in 8 was it acute. The frequency of acute delirium,

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[y] See for example ROUPELL, 1839, p. 176. [z] BARRALLIER, 1861, p. 230.
[a] BELL, 1860, ix. 38. [b] BARRALLIER, 1861, p. 82. [c] BANCROFT, 1811, p. 655.
[d] GERHARD, 1837, xx. 293. [e] JENNER, 1849, No. 2.

however, depends, in great measure, on the pursuits, habits, and constitution of the patient. In the poor and badly nourished, and likewise in the aged, whom typhus chiefly attacks, the delirium is almost always low and muttering, from the first; whereas, in the young and robust, and still more in persons in the upper class, it is often acute.

The mental state of the delirious typhus-patient is peculiar, and well worthy the study of the metaphysician. As a rule, the memory is first and most affected; judgment and power of connected reasoning often remain, after the memory has entirely gone. The mind labours under the strangest illusions, and often it appears to revolve obstinately around some fixed idea. The patients rave about objects, which have greatly engrossed their attention, either immediately preceding the attack, or years before, and which are now jumbled with persons, scenes, and events, with which they have had no connection. At other times, their ravings are centred on some article of furniture in the room, or upon their attendants, whose acts of kindness are occasionally construed into cruelty. In some cases, they are gay and jovial; in others, they pass through intense mental distress, of which a lively recollection is entertained after recovery, although, sometimes all that passes is buried in oblivion. During a few hours, some patients feel as if they had lived a lifetime; and, as a rule, time appears to the patient greatly prolonged; he almost invariably exaggerates the duration of his illness. In my first attack, my constant raving was about some rare plants, which I had gathered a few months before, on the Grampian Hills; in my second, I conceived a great dislike to my nurse, and, to a valued friend, because, on one occasion, they tied me down in bed. Somehow or other, these two individuals became mixed up with many events of my previous life; they were constantly shutting me up in dungeons from which I effected my escape; and my conviction was so firm that they intended to murder me, that on several occasions I shouted: 'Police! 'police!' I travelled in my imagination, to France, Italy, India, Burmah, and many other parts of the world, which I had really visited, trying to escape from them; but at every new place I arrived at, there these watchful demons were before me. Hildenbrand records his experience as follows: 'During an attack of 'typhus, my mind was constantly engaged in removing an awk-'ward ornament from my stove, which stood directly opposite to 'me; and being of course unable to remove it, it tormented me in 'the most cruel manner. One of my pupils, having assisted a 'short time previously at the opera, called the Mirror of Arcadia, 'performed during the whole of the nervous stage of typhus, the

'character of viper-catcher; and as he was obliged to swallow these 'disgusting reptiles, he experienced the most inexpressible anxiety. 'Another patient laboured under the painful and fantastic idea, 'that he was not only suffering for himself, but for all his com-'rades in the ward.'[f] Dr. Pickels, in his account of one of the great Irish epidemics at Cork, observes: 'A cowherd, who had 'come from the country, fancying those patients who lay around 'him were the animals, whom he had been accustomed to attend, 'endeavoured at intervals to rouse them into motion, by a particu-'lar cry, which is usual for this purpose in the country. A thief 'raved of his thefts and accomplices. A faithful steward refused, 'with many acknowledgments, to take his wine, as he had his 'master's keys, and it might render him unfit to perform his 'business.'[g] Jacquot states that one of his patients chaunted vespers for hours at a time, and also preached a sermon of an hour's length, which the nurse could follow with tolerable ease; another fought with the Russians; another gave commands to his troop; another fancied that he was the King of Spain and the Bishop of Lyons; another burst into laughter when spoken to, and was constantly epxressing his desire to go to sleep with the coffee-mill; in two instances there was hydrophobia, although in other respects, the patients were rational; while two other patients, both medical men, fancied that they were each subdivided into two persons, one of whom was in good health, and commiserated the unfortunate lot of the other, who was ill.[h] Roupell mentions the case of a female, who, for ten days, believed that she was dead, and refused to speak, except to request that she should be buried.[i]

My friend, Dr. Gueneau de Mussy, has favoured me with the following interesting account of his sensations, during an attack of typhus, caught on a visit to Dublin in 1847.

'I first imagined that I had committed a murder in France, and 'that I had made my escape to England. Extra-tradition, however, 'had been granted against me, and having the power of flying, I 'soared through the air, uttering dreadful screams and trying to con-'ceal my face with my arms and hands, in my endeavour to escape 'from a party of soldiers, who were pursuing me in a balloon and 'firing at me. I afterwards ascertained from the records kept by my 'medical attendants, that whenever I could escape from them, I 'ran about the house, with screams and gestures indicative of pro-

[f] Hildenbrand, 1811, p. 72.
[g] See Bartlett, 1856, p. 190.
[h] Jacquot, 1858, p. 190.
[i] Roupell, 1839, p. 173.

'found terror. The explanation of all this was, that the day before my 'confinement to bed, I heard of a murder committed by a gentleman 'on his wife, and that on the morning of the same day, I had wit- 'nessed the ascent of a balloon carrying four soldiers. I substituted 'myself for the murderer, and the armed men in the balloon for 'the soldiers ordered to take him in charge. Then my delusion 'took another turn. I imagined that I was tied down in bed, and, 'though feeling no pain, I believed that I was gradually being 'consumed by spontaneous combustion, while some young women, 'dressed as opera-dancers, were taking water from a pond near my 'bed and pouring it over me. With rhythmic movements, as my 'own destruction was going on, my sight grew confused, and my 'last thought at this time was, that my brain was being consumed. 'This condition probably corresponded to another period of three 'days, during which I appeared to my attendants to be quite uncon- 'scious. These illusions were interrupted by others, of a more tran- 'sient nature. For instance, at the time I was being consumed by 'fire, I saw distinctly the façade of a friend's house at Paris, in a 'state of phosphorescence, and one of his children suspended by the 'neck from a window. Another friend I saw killed in the street; and 'so strong was this last impression, that during my convalescence, 'notwithstanding assertions to the contrary, I often repeated that 'this friend was dead, and felt great concern about his loss. On 'my return to Paris, I made a point of seeing him immediately, in 'order to be convinced that he was alive. Sometimes I mis- 'took my attendants for other persons who were absent; and, after 'my recovery, I offered my thanks to a lady of Dublin, whom I 'believed to have been one of my nurses. But, during this 'delirium, I was not altogether unconscious of certain circumstances 'that occurred, and which are still fresh in my memory. Thus I 'remember, I may say I can hear, my poor friend Dr. Oliver 'Curran (who died shortly after of typhus which he caught at my 'bed-side), reading the Scriptures, and I felt comforted by his 'brotherly love.'

5. *Wakefulness*, *Somnolence*, *Coma-vigil.* During the first two or three days, the patient is sometimes heavy and drowsy, but usually until about the tenth day, there is more or less wakefulness, at all events at night. The sleep is broken and disturbed, or, for several nights, there may be none. This wakefulness may persist throughout the disease; and the first sign of amendment may be the patient falling into a quiet natural sleep. I have noted wakefulness to a greater or less extent, in 78 out of 92 cases. It is well to add, that a patient not unfrequently awakes from a sleep of several hours' duration, and insists that he has never closed his eyes, and may

dispute the point with some vehemence, although in other respects perfectly rational. This condition is the *coma-vigil* of Chomel[k] and some other writers.

But in most cases (in 57 of 92), after a period of wakefulness and nervous excitement, or occasionally without any wakefulness preceding, the patient sooner or later, but usually about the middle of the second week, falls into a state of somnolence, more or less profound. He lies on his back quiet and motionless, and with eyelids closed; if spoken to, he opens his eyes and attempts to put out his tongue, and immediately relapses into his former lethargy. As a rule, from which there are few exceptions, this state of somnolence is preceded by more or less delirium.

In grave cases, somnolence may pass into complete coma, which usually, after a few hours, or sometimes days, terminates in death. Patients, however, do often recover after having been for several days in a state of profound somnolence approaching to coma, from which it is impossible to rouse them in the slightest. Now and then, coma makes it appearance suddenly and unexpectedly, without any antecedent somnolence, and then it will usually be found that the urine is albuminous, scanty, or even suppressed.

There is another condition to which the term *coma-vigil* is more appropriately applied, but which differs from the coma-vigil of Chomel, in having the most ominous import. According to Dr. Jenner's definition, this is that peculiar condition, in which the patient lies with his eyes wide open, gazing into vacuity, his mouth partially open, his face pale and devoid of expression: the pulse rapid and feeble, or imperceptible: the breathing scarcely perceptible: and the skin cold and bathed in perspiration. He is evidently awake, but he is indifferent and absolutely insensible to all going on about him. This condition may, or may not, supervene upon somnolence; it is invariably fatal. In 9, or in more than one-fifth of Dr. Jenner's 43 fatal cases, coma-vigil was observed from one to four days before death.[l]

6. *Prostration.* Loss of muscular strength is one of the earliest and most characteristic features of typhus. In almost every case, there is more or less prostration from the first, the patient being at once struck down, so to speak, by the disease. This early and great prostration has been insisted on by all who have had much experience in true typhus. Pickels, in his report of an epidemic at Cork, observed: 'The debility was such that the patient was 'unable from the first to rise from the bed or to walk without

[k] Chomel, 1834. [l] Jenner, 1849, No. 2.

'assistance, and in some instances, even without the effort of 'rising, fainted in bed.'[1]

On the second or third day of the disease, the patient is compelled to take to bed, and before the end of the first week, he is usually brought to hospital. Of 64 cases, I ascertained that the patients took to their beds on the first day in 22, on the second day in 28, on the third day in 10, on the fourth in 2, and on the sixth in 2. Again, of 76 patients, 1 had been ill only one day before admission into hospital, 7 two days, 2 three days, 8 four days, 15 five days, 5 six days, 13 seven days, and 25 more than a week. Thus, 33 cases (or 43·4 per cent) had not been more than five days ill, and 51 cases (or 67 per cent) not more than seven days. The mean duration of all the cases before admission was $7\frac{1}{3}$ days. Of 149 cases under Dr. Craigie at Edinburgh, 125 (or 84 per cent.) were admitted into the Infirmary on, or before, the eighth day.[m] Of 27 fatal cases recorded by Jenner, all were confined to bed by the sixth day.[n]

As a rule, the prostration increases as the disease advances, until about the tenth or twelfth day, when it is extreme, the patient being perfectly helpless, and unable to assist himself in any way. Out of 90 cases, I noted this extreme prostration in more than one-half. In 34 of Dr. Jenner's 43 fatal cases, this extreme prostration was noticed, and in most it came on from the ninth to the twelfth day of the disease.

The prostration is always very great in those cases where there has been violent delirium, the strength being exhausted by the extraordinary efforts called into play during the stage of excitement.

Sometimes there appears to be little loss of strength during the first six or eight days of the disease, and then extreme prostration sets in suddenly, and may prove rapidly fatal. This form is chiefly observed in persons who have struggled against the disease for several days, and followed their ordinary avocations, and hence the importance of husbanding the strength from the first.

In most cases, the patients are not only weak, but complain from the first, of a *feeling* of great weakness and lassitude.

7. *The Decubitus* is in most cases dorsal. Except where there is restlessness and active delirium, the patient lies on his back, with his arms extended along the chest, and the forearms slightly flexed, the hands resting on the hypogastric region and sometimes interlaced. As the prostration increases, the head sinks from the

[1] Bartlett, 1856, p.196. [m] Craigie, 1837, No. 2, p.328. [n] Jenner, 1849, No. 2.

pillow, and the whole body gravitates towards the bottom of the bed.

8. *Muscular Paralysis.* In addition to the general loss of power in the muscular system, there are certain muscles, which often become entirely paralysed about the tenth or twelfth day. In most severe cases, there is paralysis of the neck of the bladder and of the sphincter ani, causing involuntary discharge of urine and fæces. The urine constantly dribbles away, soaking the bed clothes and irritating the skin. At other times, owing to paralysis of the coats of the bladder, there is retention of urine, and recourse to the catheter is necessary. It must not be forgotten, that retention and incontinence may co-exist, the urine dribbling away from an over-distended bladder. Consequently, in all cases of typhus, with great nervous prostration, the physician must not be satisfied by being told that the patient makes water, but must examine the hypogastric region daily, by palpation and percussion. Out of 90 cases, I found that the stools and urine were passed involuntarily in 18, and the urine only in 29, while in 5 cases there was retention of urine. Of 50 cases, in which there was involuntary discharge or retention of urine, 10 died; while, of 40, where these symptoms were absent, only 1 died. Of Dr. Jenner's 43 fatal cases, there was retention or involuntary discharge of urine in upwards of one-half, and involuntary discharge of fæces in 17 cases.

The meteorism already alluded to, the occasional dysphagia, the inarticulate speech or complete aphonia, and the inability to protrude the tongue, all indicate paralysis of different parts of the muscular system. Of these symptoms, the worst is dysphagia, which is usually the forerunner of death.

Occasionally, the orbiculares muscles appear to be paralysed: the patient is unable to close his eyelids, and ulceration and sloughing of the cornea sometimes result from the constant exposure.

9. *Muscular Agitation.* In few severe cases, is some degree of tremulousness of the hands and tongue not observed during the second week. Occasionally, the entire body is in a constant state of tremulous agitation, which is increased when the patient is spoken to, or in any way excited. Of 90 cases, I have noted great tremulousness in 12. The symptom is most developed in the aged and infirm, or in persons who, previously to their attack, have been much addicted to spirituous liquors, or been subjected to mental labour. It always indicates great prostration. (See page 153).

Subsultus tendinum and spasmodic twitchings of the face are observed in many severe cases. The tendons at the wrist are those most frequently affected. When the twitchings attack the face, one angle of the mouth is usually drawn up. Dr. Jenner alludes to two instances, in which the spasmodic action of the inferior recti muscles of the eyes, and of the levatores palpebrarum, gave a peculiar aspect to the countenance; in both cases, the movements were excited at any moment, by suddenly raising either arm.

In one of Barrallier's cases, there were well-marked choreic convulsions.[o]

Another modification of these spasmodic movements is picking or fumbling with the bed-clothes, or what is called *Floccitatio* or *Carphology*. The hands are extended in every direction, above the head and outside the clothes, while prehensile movements are exercised with the fingers, as if the patient desired to draw towards him some imaginary object.

Obstinate hiccup, often associated with great meteorism, is another symptom occasionally met with.

All of these symptoms are of grave import, particularly subsultus, carphology, and obstinate hiccup. I have known patients however, recover, notwithstanding the occurrence for several days of subsultus, carphology, and general tremors.

10. *Muscular Rigidity.* Contraction and rigidity of certain muscles are observed more rarely, and only in severe cases. The fingers may be tightly clenched, or the fore-arms flexed, or in rare cases there is trismus or strabismus. In several cases, I have observed tonic spasms of many different muscles; twice I have seen the legs and thighs so bent that the knees almost touched the chin; both patients died. M. Godélier observed well-marked catalepsy in one case, a female, at the hospital of Val de Grace,[p] and a similar case, also a female, has come under my notice.

11. *General Convulsions* constitute one of the most formidable symptoms of typhus. All writers, in fact, since the time of Hippocrates, have regarded convulsions as an almost fatal symptom in fever.[q] The cases of typhus, where they occur, are almost invariably fatal, unless the patient has previously suffered from epilepsy.[r] Dr. Henderson, however, mentions the case of a boy aged 14, who recovered: after several days of stupor this boy was

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[o] Barrallier, 1861, p. 83.

[p] Godélier, 1856, p. 893.

[q] Hippoc. *Aph.* iv. 66, 67; also Graves, 1848, i. 240.

[r] Instances have been known where epileptic fits were suspended during typhus. (See G. A. Kennedy, 1838, p. 22).

seized with convulsive movements of the upper and lower limbs, insensibility and strabismus; the fit lasted for about an hour, and did not recur.[s] Another case of recovery, after two severe fits of convulsions, is recorded by Dr. Hudson; in this case, the treatment consisted in abstracting ten ounces of blood by cupping from the neck, and purging with calomel.[t] A third case is reported by Graves,[u] and three others have come under my own notice. In Case XI., convulsions came on about the fourteenth day, and recurred several times daily for nine days, the intervals between the fits being characterized by great rigidity of the muscles and almost complete unconsciousness; but the patient recovered.

The convulsions do not appear until an advanced stage of the fever, usually about the middle or end of the second week. Of 19 cases recorded by Christison,[x] Hudson,[y] Graves,[z] G. A. Kennedy,[a] Aitken,[b] Jenner,[c] Steven,[d] Todd,[e] and G. Johnson,[f] they appeared on the seventh day, in 1; on the ninth day, in 3; on the tenth day, in 3; on the eleventh day, in 3; on the twelfth day, in 5; on the thirteenth, in 1; on the fourteenth, in 2; and on the fifteenth day, in 1. Of 6 cases, under my own care, where the duration of the fever was known, convulsions occurred on the tenth day, in 1; on the thirteenth, in 2; and on the fourteenth, in 3. The fit is usually preceded, for a day or two, by an unusual amount of drowsiness or delirium; but in some cases the previous symptoms have been mild, or convalescence may seem to have commenced. In most cases, where attention has been directed to the circumstance, the urine has been found scanty: in one of Dr. Christison's cases, the quantity, for four successive days prior to the attack, was only 16, 12, 8, and 3 ounces. Death takes place either immediately after the first fit, or within two or three days, but, in most cases, in less than twenty-four hours. The convulsions are usually followed by coma, which continues until death, and may, or may not, be interrupted by a recurrence of the paroxysms.

No appearance is ever found within the head, to account for the convulsions. Dr. Jenner records a case, in which a film of extravasated blood was found after death in the cavity of the arachnoid, over the convex surface of the anterior lobe of the left hemisphere, but he was inclined to regard it as the result, rather than the cause,

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[s] Henderson, 1839. [t] Hudson, 1837, p. 353. [u] Graves, 1848, i. 239.
[x] Christison, *On Granular Degeneration of the Kidneys*, 1839, p. 167.
[y] Hudson, 1837, pp. 344, 353; and 1842, p. 282. [z] Graves, 1848, i. 239.
[a] G. A. Kennedy, 1837. [b] Aitken, 1848. [c] Jenner, 1850, xxi. p. 15.
[d] Steven, 1855. [e] Todd, 1860, p. 143. [f] Johnson, 1862.

of the fits.[g] The same lesion has been repeatedly observed after death from typhus, where there have been no convulsions, (see *Anatomical Lesions*), and its occurrence in cases of convulsions is entirely exceptional. Moreover, hæmorrhage in this locality would be more likely to cause coma than convulsions. The convulsions cannot be attributed to the pressure of intra-cranial fluid, for, in many of the cases, the quantity of this fluid has been unusually small,[h] and in many cases where there is abundance of fluid, there are no convulsions.

It is now tolerably certain, that convulsions occurring in the course of typhus, have always a uræmic origin. In most cases, there is evidence of obstruction to elimination by the kidneys, in the presence of albumen in the urine. In 8 out of 9 cases, where the urine was examined by myself, albumen was present in greater or less quantity. Dr. Christison states that in every case of typhus that has come under his notice, and been submitted to proper investigation, convulsions have been connected with an albuminous state of the urine, and organic disorder of the kidneys.[i] In Dr. Todd's case, the urine was albuminous, and contained blood-casts.[k] In Dr. Johnson's case,[l] the urine was scanty, dark, like porter, and highly albuminous; and the patient was recovering from acute Bright's disease, at the time of his seizure with typhus. In one case, Dr. Christison discovered urea in large quantity in the serum of the blood, and this observation was verified in Case X. Frerichs also has shown, that convulsions, occurring in the course of any of the eruptive diseases, are connected with the presence of albumen and casts in the urine, and of urea, or carbonate of ammonia, in the blood.[m]

But although albuminuria is the rule, it is not invariably present when convulsions occur in typhus. In two of my cases the quantity of albumen was extremely small; and in one, none could be discovered. Still the absence of albumen, and even an apparently healthy condition of the kidneys, are not opposed to the theory of uræmic convulsions. Abundance of urea has been found in the blood of relapsing fever, complicated with convulsions, where the urine was non-albuminous, and the kidneys apparently healthy.[n] Disease of the kidneys merely increases the chances of convulsions occurring, by impeding the excretion of urea.

Moreover, while it is well known that albuminuria often exists

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[g] Jenner, 1850, xxi. 15.
[h] See 2 cases mentioned by Peacock, 1843.
[i] *Op. cit.*, p. 171.
[k] Todd, 1860, p. 143.
[l] Johnson, 1862.
[m] *Die Brightsche Nierenkrank.* 1851.
[n] See remarks on the Urine and on Convulsions, in Relapsing Fever.

in typhus, where convulsions never appear (see page 148), it is also probable, that urea and other products of tissue-metamorphosis, which ought to be eliminated by the kidneys, are often accumulated in the blood, independently of convulsions, and account for the stupor and other symptoms of the typhoid state.

The appearances presented by the kidneys, after death from convulsions in typhus, vary. Sometimes, there is unmistakeable evidence of disease of old standing, the organs being hypertrophied and fatty, or atrophied and granular; but, not uncommonly, the morbid appearances are evidently recent, and secondary to the fever. Occasionally, as in Case X., the kidneys present the characters of acute nephritis. At other times, with the exception of moderate hyperæmia, they appear healthy; but, on careful examination, the cortex is found slightly hypertrophied and friable, and the uriniferous tubes are gorged with epithelium cells, containing a quantity of minute granules. In two cases of convulsions examined by Jenner, the kidneys were said to be healthy.[o]

The following cases are examples of convulsions occurring in typhus.

Case VII.

Typhus of a mild form. On evening of fourteenth day, after apparent improvement, sudden seizure with Convulsions, followed by Coma, and Death on the sixteenth day. Autopsy:—Slight subarachnoid serosity, but Brain and Membranes otherwise normal. Kidneys hypertrophied, granular, and fatty.

Mary H——, aged 45, admitted into London Fever Hospital, August 7th, 1856; ill eight days.

The symptoms were:—pulse 120; severe headache; pains in the back and limbs; great restlessness, and want of sleep, but no delirium; skin hot and dry, with a distinct typhus rash on the chest and abdomen; tongue brown; great thirst; bowels confined; occasional cough, with mucous expectoration. On the fourteenth day, the rash had disappeared; the pulse had fallen to 86, the tongue was cleaner, and the appetite was returning. During the day, however, the urine was very scanty, and in the evening, the patient was suddenly seized with insensibility, convulsive movements, and foaming at the mouth, followed by profound coma and contracted pupils. On August 16th (sixteenth day), she was still unconscious and motionless; pupils rather large and insensible to light; took no notice when hands were pinched; breathing stertorous and irregular, and occasionally suppressed for a few seconds; pulse almost imperceptible; stools and urine in bed; skin bathed with perspiration. Died on evening of 16th. The treatment consisted in sinapisms to the neck, blisters to the scalp, a purgative, and stimulants.

[o] Jenner, 1850, xxi. 15.

Autopsy, 43 hours after death.

Arachnoid transparent; pia mater moderately injected; no extravasation of blood; membranes separate readily from brain; small quantity of subarachnoid serosity between the sulci; no lymph, pus, or tubercle. Substance of brain firm, but normal; no bloody points visible on section of white substance; cortex rather dark; choroid plexuses pale; less than half a drachm of fluid in each lateral ventricle.

Half an ounce of clear serum in pericardium; muscular tissue of heart pale and rather soft; valves and vessels normal. Extensive emphysema at apices of both lungs; a moderate amount of frothy secretion in bronchial tubes; slight pulmonary hypostasis.

Stomach and intestines healthy. No abnormal appearance of Peyer's patches or of the solitary glands of the ileum. Liver 3 lbs. avoird.; texture friable and lobules indistinct; an ounce of dark, viscid bile, in gall-bladder. Spleen 6 ounces, rather soft. Right kidney, $5\frac{1}{2}$ ounces; left, $5\frac{3}{4}$ ounces; surfaces of both kidneys distinctly granular; cortical substance hypertrophied, pale, yellow, and friable, with several cysts, about the size of a pea, near outer surface; secreting cells loaded with oil. Bladder empty.

Case VIII.

Typhus. Attack of Convulsions on thirteenth day, followed, in thirteen and a half hours, by Death. Autopsy:—Brain and Membranes healthy. Hypostatic Congestion of Lungs. Old Disease of the Kidneys.

Elizabeth W——, aged 49, admitted into London Fever Hospital January 17th, 1857. Eight days before, had been seized with headache, general pains, and lassitude.

January 18th (tenth day). Pulse 120, weak. Tongue moist, with dirty, brownish fur. Faint typhus-eruption on skin. No headache; sleeps at intervals; but expression very stupid, with some delirium. Camphor mixture, wine (4 ounces), and beef-tea, were prescribed.

January 19th (eleventh day). Pulse 120; prostration increased; tongue dry and brown; three stools, not in bed. Skin cool; eruption more abundant, darker, and partly petechial. Slept badly, and has been very restless and delirious. Mental faculties are very dull and confused, and there is great deafness. Pupils contracted. Brandy (four ounces) was ordered, in addition to the wine.

January 21st (thirteenth day). About 12·30 a.m., patient was suddenly seized with convulsions and foaming at the mouth. The bowels were opened four or five times yesterday, but the urine has been very scanty. The convulsions lasted for a few minutes, and did not return, but were followed by profound coma, which lasted till death, at 3 p.m. The muscles of the right arm were rigid; the left angle of the mouth was drawn up; the pupils were dilated and insensible to light. The respirations were noisy and blowing, and the pulse was scarcely perceptible. Little or no urine was passed, and there was no dulness or tenderness over the pubes.

Autopsy, 20 hours after death.

Typhus-eruption visible on skin. Upper and lower extremities rigid. No œdema.

No increased vascularity of membranes of brain. Sinuses moderately filled with fluid blood. No extravasation. Very scanty sub-arachnoid serosity on under surface of middle lobe of brain. No fluid in lateral ventricles. Brain-substance normal.

Pericardium contained 1½ ounce of clear serum. Heart soft and flabby; right cavities filled with dark, fluid blood. Both lungs infiltrated with serous fluid, and much condensed posteriorly, each weighing about 36 ounces avoird. The condensed portions were non-granular, on section.

The liver weighed 2½ lbs.; its tissue was soft, flabby, and friable, and presented a pale nutmeg appearance; the secreting cells contained an unusual amount of oil. The kidneys were small; left, 3½ ounces, and right, 3¼ ounces; surfaces marked by large granulations, and capsules firmly adherent; cortical substance atrophied and dense, with several cysts. Intestines perfectly normal.

Case IX.

Typhus. Delirium ferox, followed by Convulsions, Coma, and Death. Urine albuminous, with casts of the uriniferous tubes. Autopsy:—Moderate amount of sub-arachnoid serosity, but Brain and membranes otherwise healthy. Recent disease of the Kidneys.

Richard H——, aged 40, admitted into the London Fever Hospital, on March 5th, 1862, at 3 p.m., having been ill about ten days. On admission, the patient was in a state of acute delirium, shouting loudly, and was with difficulty kept in bed. The face was flushed, the conjunctivæ injected, and the pupils contracted. There was a copious well-marked typhus-rash; pulse 120, full and soft. The patient had not been half an hour in bed, before he had several attacks in rapid succession of convulsions, with opisthotonos and foaming at the mouth. After the fits, he continued restless for some time; but in a few hours, he passed into a state of coma, which lasted until death, at 6 a.m. the following morning. The treatment consisted in shaving the head, a blister to the scalp, a drop of Croton oil by the mouth, and a draught every three hours, containing nitric ether (℥j), and acetate of potash (ʒj).

Autopsy, 36 hours after death.

No œdema of integuments. Moderate vascularity of membranes of brain. Small quantity of sub-arachnoid serosity; six drachms of serum at base; less than half a drachm in each lateral ventricle. No extravasation. Brain-substance normal.

Heart flabby and somewhat soft, but muscular fibres, under microscope, apparently normal. Small, dark friable coagulum in right ventricle; but blood mostly fluid and dark. No staining of lining membrane of heart or vessels. Old adhesions and false membrane on surface of right lung; moderate hypostatic congestion of both lungs.

Intestines normal. Liver 54 ounces, pale-yellow, smooth and friable; the hepatic cells contained an increased amount of oil. Spleen 8 ounces, diffluent. Both kidneys much enlarged; left, 8½ ounces; right, 8 ounces; smooth and rather pale; capsules separated readily. Cortical substance hypertrophied, and contained a few cysts, up to the size of a pea. All the uriniferous tribes were gorged with epithelium cells, which appeared filled with minute granules, and a few oil-globules; the amount of oil was far from being sufficient to constitute a 'fatty kidney.' The bladder contained 4 fluid-ounces of urine, which had specific gravity of 1010, and contained a considerable amount of albumen. A copious flaky deposit separated on standing, composed of renal and vesical epithelium, and numerous hyaline, and epithelial, casts of the uriniferous tubes. The renal epithelium cells, both free and entangled in the casts, were similar to those found in the kidneys.

Case X.

Typhus terminating in Convulsions and Death on the ninth day. Autopsy: Acute Nephritis. Blood, fluid, and containing Urea.

Emma C——, a robust female, aged 32, was admitted into the London Fever Hospital, April 25th, 1862, her illness having commenced six days before, with shivering, pain in the limbs, and headache.

On admission, pulse 84, and feeble; skin, warm and dry; typhus-rash well out; tongue dry in the centre; bowels open; stupid and confused, and rather drowsy; pupils small. Beef-tea, milk, wine (6 ounces), and carbonate of ammonia were prescribed.

The patient continued much in the same state, and there was nothing to excite alarm, except that she was a little more drowsy; still, she always answered, when spoken to. But at 10 p.m., of April 27th, she was suddenly seized with violent convulsions and foaming at the mouth, followed by death at 10½ p.m. Her bowels had been open, the same morning; but the nurse could not be certain, whether she had passed water.

Autopsy, 17 hours after death.

Slight rigidity; perceptible pitting of lower extremities, on pressure; typhus-spots still visible on chest and abdomen.

Sinuses of brain filled with dark, fluid blood; moderate vascularity of pia mater. A small amount of sub-arachnoid serosity; two drachms of serum at base, and one drachm, in each lateral ventricle. Brain-substance normal.

An ounce of clear serum in pericardium. Right cavities of heart, and large veins, filled with dark, fluid blood; muscular tissue and valves normal. A few ounces of serous fluid in both pleural cavities, and moderate hypostatic condensation of both lungs.

Peyer's patches, and the solitary glands perfectly normal. Liver, hyperæmic; spleen, 7½ ounces, pulpy. Both kidneys were much enlarged: left, 6¾ ounces; right, 6½ ounces. The capsules separated readily, and the surfaces were smooth; but both organs were of an intensely dark, chocolate

colour, darker even than those figured by Bright (*Reports*, vol. i., Pl. V.) The outer surface was marked by a number of little rounded dots, of a still darker hue. The consistence was firm; a quantity of blood dripped away on section. The tubes were gorged with renal epithelium, and many of them contained blood. Not a drop of urine in bladder.

Three ounces of blood, from the right side of the heart, were shaken for some time, with six ounces of alcohol, and then filtered. The filtered fluid was slowly evaporated to dryness, on a sand-bath. The residue was dissolved in two ounces of alcohol, warmed, and filtered. The filtered fluid was a second time evaporated to dryness, and the residue, treated with two ounces of distilled water. After filtration, this fluid was evaporated to the consistence of syrup, and then treated with half its volume of nitric acid. Slight effervescence occurred, and a large number of crystalline scales, presenting the characteristic rhomboidal form of nitrate of urea, were formed. A decided urinous odour was given off during evaporation, and after the addition of the acid.

Nitrate of urea was also obtained, in smaller quantity, from the blood contained in the sinuses of the dura mater, by the same process.

Case XI.

Typhus. Epileptiform Convulsions commencing on thirteenth day, and recurring repeatedly, for nine days. Albuminous urine. Recovery.

Isaac T.—, aged 17, was admitted into London Fever Hospital, April 12th, 1862. His father, mother, and brother had all had typhus fever, and one person, with the characteristic eruption, had been brought to the hospital from the same house, shortly before. He never had fits of any sort, except one in infancy, during dentition. His left leg had been amputated, some years before.

Twelve days before admission, he had been taken ill with shivering, headache and loss of appetite; after a few days, according to his mother, he became spotted all over; and for a week before admission, he had been violently delirious. The man, who brought him to the hospital, stated, that he had 'a fit' during the journey. On admission, he was extremely restless and delirious, raving about his purse and looking for imaginary objects under the bed. The pulse was 84, and feeble; tongue moist and slightly furred; bowels open. At 2 p.m. of April 13th, patient had a fit of convulsions, lasting for nearly half an hour, followed for an hour by slight stupor, and then by a return of the delirium. Beef tea, milk, and a mixture of nitro-hydrochloric acid and nitrate of potash, were prescribed.

April 14th (15th day). Last night was violently delirious, but slept soundly for several hours after two doses of Vin. Ant. Pot. Tart. (♏ xx.), and Liq. Morph. Acet. (♏ x.). This morning had two more fits, each lasting for half an hour. At 2p.m. was very restless and delirious, with contracted pupils and great rigidity of muscles of arms. Pulse 100 and feeble; no rash; 3 motions. Urine partly passed in bed;

specific gravity 1010; clear, and contains a considerable amount of albumen. Head to be shaved and blister to scalp. Wine 4 ounces.

April 15. Two more fits. Urine still albuminous, with a very copious deposit of colourless, rhomboidal crystals of uric acid; specific gravity 1013.

April 16. Pulse 80. Three fits since yesterday. Eyes, at time of visit, staring and fixed; pupils natural; scarcely conscious, but took notice, when spoken to. The muscles of arms were so rigid, that the entire body was raised, in the attempt to extend them. Urine still albuminous, and depositing lithic acid. No œdema. The blister to the scalp was repeated.

On April 17, the albumen had disappeared from the urine and the patient was ordered an egg daily, and iodide of potassium (3 grains) three times a day, which was taken for eight days.

The patient continued in the same state, with two fits daily, up to the morning of April 21st. After this, the fits did not recur, and all the other symptoms improved; on the 25th April, the patient was allowed meat; and on May 12th, he left the hospital, in his usual health.

Case XII.

Typhus. Slight Convulsions about the fourteenth day, followed by Stupor, and scanty, Albuminous Urine. Return of convulsions, on the 17th and 20th days. Death, on the 22nd day. Autopsy:—Large amount of serous fluid beneath the arachnoid, and in the lateral ventricles. Recent disease of the Kidneys.

John B ——, aged 21, was admitted into the London Fever Hospital, April 22nd, 1862, about the ninth day of an attack of typhus. On admission, pulse 120; tongue dry and brown in centre; bowels confined; skin warm and dry, and covered with a copious petechial typhus rash; very confused; sleeps badly; occasional delirium; contracted pupils. Urine 1018, rather dark, free from albumen. Wine and brandy (4 ounces of each daily), a dose of castor oil, and a mixture containing carbonate of ammonia, were prescribed.

On the 25th, pulse 120, and feeble. Very restless and delirious last night, but slept after a draught containing 10 minims of Liq. Morph. Acet. Urine free from albumen.

On the 26th, pulse 120; was quieter, but very drowsy, and with difficulty roused. Urine very scanty, and contained a small amount of albumen. Rash fading.

On the morning of the 27th, the patient had a fit of convulsions, with foaming at the mouth, lasting for a few minutes. After the fit, pulse 72, and feeble; pupils of normal size, but insensible to light; was very deaf and almost unconscious.

The patient continued in a drowsy, stupid state, for three days, the pulse not exceeding 72, and the urine containing albumen every day, except on April 28th. Meanwhile, the rash entirely disappeared.

On the evening of April 30th, had another fit, and on the following day

pulse 120; tongue moist, very drowsy; urine again very scanty, and contained a large amount of albumen.

A third fit occurred on the evening of May 3rd. After this, the urine became more copious, the albumen disappeared and did not return, and the patient seemed a little more conscious; but the pulse rose to 140; the tongue became dry and brown; and the prostration rapidly increased, until death on May 5th.

The treatment, after the fits, consisted in blisters to the scalp, sinapisms over the kidneys, and enemata to open the bowels.

Autopsy, 48 hours after death.

The only abnormal appearance, within the cranium, was a large amount of subarachnoid serosity, with two drachms in each lateral ventricle, and about an ounce at the base.

Hypostatic congestion of both lungs. Right cavities of heart contained a small pale coagulum, and black fluid blood; left cavities filled with dark coagulum.

No trace of disease, in Peyer's patches, or in the solitary glands of the ileum. Liver flabby and friable. Spleen $7\frac{1}{2}$ ounces, rather soft. Both kidneys enlarged, especially left; right, $4\frac{3}{4}$ ounces; left, 7 ounces. Capsules non-adherent; surfaces smooth; pyramids considerably injected, but cortex rather pale. Uriniferous tubes gorged with epithelium.

h. Morbid Phenomena presented by the Organs of Special Sense.

1. *Organs of Vision.*—The conjunctivæ are in most cases much injected, from an early stage of the disease. Dr. Jenner noted this appearance in 25 out of 43 fatal cases. The blood in the conjunctival vessels is of a dark hue; the membrane rarely presents the bright red tinge observed in acute inflammations of the brain, or of the eye itself. Occasionally, extensive ecchymoses, of a brick red colour, are observed beneath the conjunctivæ; in one case, Barrallier found extravasations of blood between the layers of the cornea.[o] During the first week the eyes are usually moist, but afterwards they may be dry.

The pupils, in the advanced stages of severe cases, are mostly contracted, and often insensible to light. Sometimes they are contracted to a mere point—the *pin-hole pupil* of Graves. This contracted pupil may accompany active delirium, or profound stupor. I have rarely, if ever, seen dilated insensible pupils associated with typhomania, or delirium tremens in genuine typhus. A similar observation has been made by Dr. W. T. Gairdner and Barrallier.[p] Occasionally when the stupor is very profound, or is passing into

[o] Barrallier, 1861, p. 224.

[p] W. T. Gairdner, 1862, No. 2, p. 148; Barrallier, 1861, p. 79.

coma, the pupil, which before was contracted or natural, becomes dilated, and sometimes slight strabismus is observed.

Photophobia is not uncommon: it was noted by Barrallier in one-third of 1,058 cases.

2. *Organs of Hearing.*—Tinnitus aurium, and noises in the ears, of various sorts, are occasionally complained of, during the first four or five days of the disease, and again during convalescence.

Deafness, often complete, of one or both ears, is a very common symptom after the fifth day, and may persist for several days, after the commencement of convalescence. I am unable to state its precise frequency in figures; but during the present epidemic (1862), it has occurred, in a greater or less degree, in fully one-half of the cases under my care. Since the time of Fracastorius,[q] deafness has been regarded as a favourable symptom; but it is doubtful, if there be any grounds for such a belief. It is true, that many cases recover, in which there has been complete deafness; but, on the other hand, deafness is present in a large proportion of the cases which prove fatal (in one-fifth of Dr. Jenner's). Deafness, however, is favourable, when contrasted with the opposite state, or intolerance of sound, which is sometimes met with. It is difficult to give a satisfactory explanation of the deafness: it is far too common, and often too complete, to be due to accumulation of wax, or to swelling in the fauces; and it certainly is quite independent of the administration of large doses of quinine, as suggested by Barrallier. An ingenious explanation has been offered by Dr. Stokes. He thinks that the muscles of the ear, like the muscles of the body generally, become softened, so that they no longer maintain the conditions, necessary for the proper communication of the atmospheric vibrations to the inner chambers. Occasionally, deafness is accompanied by otorrhœa, and it may then be due to inflammation of the lining membrane of the meatus.

3. *Organ of Smell.*—A catarrhal state of the pituitary membrane is not uncommon, at the commencement of the disease.

Epistaxis rarely occurs at any stage of uncomplicated typhus. I have met with it in only two or three cases, and then it was always scanty, and was probably due to picking the nose. Jenner noted epistaxis in only two cases; but in one it was very slight, and in the other the patient had been liable to attacks during health. But, under certain circumstances, as when typhus is complicated with scurvy, epistaxis appears to be more common. Among

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[q] 'Surditas salutem portendit' (FRACASTORIUS, 1546). 'Deafness is rather a favourable symptom in typhus' (ALISON, *University Lect.*, 1849, not pub.)

the French troops in the Crimea, where typhus was so often complicated with scurvy, Jacquot found epistaxis in about one-fourth of the cases; it was most common in the early stage, but occasionally it seemed to be critical; the quantity was sometimes so great, as to necessitate plugging the nose.[r] Barrallier observed epistaxis in 97 out of 1302 cases, among the prisoners at Toulon, but in all, except 11, which were mostly complicated with scurvy, the bleeding was slight.[s] Many instances of 'petechial fever,' with copious hæmorrhage from the nostrils, which have been observed during some of the Irish epidemics,[t] have probably been either examples of relapsing, or pythogenic fever.

4. *Organ of Taste.*—The taste is usually perverted from the first. All articles of diet, and more especially sweet things, are thought to have a bad taste. Acids are longest relished; but after a time, cold water is preferred. In the advanced stages of severe cases, all sense of taste is usually abolished.

5. *Cutaneous Sensibility.*—Complete anæsthesia of the entire surface is sometimes met with towards the termination of grave cases, even when the patient is sufficiently conscious to give rational answers.

The opposite condition, or hyperæsthesia, is occasionally observed. The patient starts, or calls out, on the slightest touch, or movement of the bed-clothes. In the Philadelphia epidemic of 1836, Gerhard states, that the sensibility of the skin was always augmented, when the stupor was not so great as to render the patient insensible, or nearly so, to all external impressions.[u] The hyperæsthesia may be general, or partial. In the latter case, it may be confined to the abdomen, a circumstance to be remembered in diagnosis from typhoid fever, as it is then apt to be confounded with the tenderness, resulting from intestinal lesion.

Section VII.—Stages and Duration of Typhus.

Authors have divided typhus into different stages. Hildenbrand made eight; Jacquot, three: and Barrallier, five stages. Although all such divisions are entirely arbitrary, the following appears to me to be in many respects convenient, and to apply to the majority of cases:—1, the stage of Incubation; 2, the stage of Invasion; 3, the stage of Nervous Excitement; 4, the Typhoid stage;

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[r] Jacquot, 1858, pp. 180, 198. [s] Barrallier, 1861, pp. 227, 359.

[t] Profuse hæmorrhages from the nose were very common in the epidemic of 1740. (See O'Connell, 1746, and Rutty, 1770, p. 88). Many of the cases, however, were probably relapsing fever. [u] Gerhard, 1837.

5, the stage of Remission or Crisis; 6, Convalescence. The duration of these stages varies in different cases: some may be shortened, or altogether absent; and occasionally it may be difficult to say, when one stage ends, and another begins.

1. *The Period of Incubation* has been already considered (see page 89).

2 *The Stage of Invasion* extends from the commencement of indisposition, to the appearance of the eruption.

The access of typhus is usually rather sudden, as compared with that of pythogenic fever, but less, so than that of relapsing fever. It is rare for the patient or his friends, to be unable to date the commencement of the attack. The patient is seized with cold shivers, lassitude, and disinclination for exertion, followed by pains in the limbs and back, headache, loss of appetite, white tongue, and thirst. Most commonly, there are no marked rigors, but merely a feeling of chilliness, for the first two or three days, so that the patient is unwilling to leave the fire. In some cases, the first symptoms are those of slight febrile catarrh. Occasionally, though rarely, the symptoms above mentioned are accompanied by nausea and sickness. Of 30 cases, in which I have noted the symptoms of the disease at its commencement, in 22 it began with cold shivers or chilliness, and lassitude, followed by pains in the limbs and headache; in several of the 22 cases, there was also slight catarrh; in 8 cases, there were no rigors or chilliness at first, but the disease commenced with pains in the limbs and headache. The above symptoms were associated, in 2 cases, with nausea and sickness, and, in 4 cases, with great drowsiness; in 1 there was delirium on the first night, and in 1, there was slight sore throat. In 6 of the 30 cases, the chills, or pains in the head and limbs, were preceded, for some days, by premonitory symptoms, such as lassitude and disinclination for exertion, vertigo, loss of appetite, or febrile catarrh, with much prostration; in the remainder, the patient had previously been in perfect health. When premonitory symptoms occur, there may be some difficulty in fixing the precise date of the commencement of the disease, although this is usually marked by the sudden accession of headache, rigors, or chilliness. The premonitory symptoms can scarcely be regarded as part of the fever, first, because they are in most cases absent; and secondly, because nurses and other attendants on the sick, often complain of similar symptoms, without typhus succeeding. It is not impossible that, as Jacquot suggests, they are sometimes due to 'une typhisation à 'petite dose, au milieu de laquelle survient le vrai typhus.'[x] In

[x] Jacquot, 1858, p. 162.

other cases, a febrile catarrh may have been the predisposing cause of the typhus. In cases, where the eruption has been said to appear later than the seventh day, premonitory symptoms have probably been included, in reckoning the duration of the disease.

3. *The Stage of Nervous Irritation* usually extends from the appearance of the eruption, until the commencement of somnolence, and is characterized by restlessness, sleeplessness, and delirium. During this stage, the headache ceases, and the tongue begins to grow dry and brown.

4. *The Typhoid, Putrid, or Malignant Stage* is characterized by extreme prostration, great impairment of the intellect, low muttering delirium, stupor, and more or less unconsciousness, sometimes passing into coma; not uncommonly, involuntary evacuations, tremors and subsultus; sordid teeth, dry, brown, crusted tongue; and rapid, small, soft pulse. It is not every case of typhus, that presents this stage; but the earlier, and more marked, the 'typhoid state' is developed, the more severe is the case.

Many other diseases besides typhus—other idiopathic fevers, blood-poisonings, and local inflammations—often pass into the 'typhoid-state.' In other words, they come to resemble typhus, by presenting a group of symptoms of which it is considered the type. The early, and some modern writers, speak of a case, which assumes such characters, as *putrid* or *malignant*. Pythogenic fever, malarious remittent fever, yellow fever, cholera, uræmia from kidney-disease, pyæmia, acute phthisis, and pneumonia are familiar examples of diseases, occasionally assuming a typhoid, malignant, or putrid character. Although, in some cases, especially when there is no local lesion, it might be difficult to distinguish the typhoid state induced by one disease, from that induced by another, this difficulty affords no more ground for arguing, that all Continued Fevers are identical in origin, than for maintaining, that typhus exists in every disease that assumes the typhoid state. It is the fashion with some, indeed, to speak of typhus, and the 'typhoid-state' as synonymous, and thus we commonly hear of cases of 'gastric fever' or diphtheria, 'passing into typhus.' But true typhus has a mode of origin, and a clinical history of its own, which do not admit of its being confounded with every disorder, which assumes a 'typhoid state.'

It is very possible, however, that the typhoid state may have a common origin in all diseases, or may be due to the accumulation in the blood, of the products of disintegrated tissue, as the result of the primary malady. The chief of these products are probably

urea and carbonate of ammonia. When these products are retained in the system, in consequence of organic disease of the kidneys, a condition is induced, which it would be often impossible to distinguish from the typhoid stage of typhus. In the typhoid stage of cholera, it is well-known, that there is a remarkable retention of the urinary solids in the blood. Again, in malignant (or typhoid) cases of yellow fever, Roche has found large quantities of urea in the blood;[y] Blair has detected a large amount of carbonate of ammonia in the blood, and also in the expired air; while Lallemant describes the sweat, as of a penetrating, urinous odour.[z] So also in typhus, urea has been found in the blood, the breath contains a great excess of ammonia, the skin has often an ammoniacal odour, and the stools are occasionally ammoniacal, and loaded with crystals of ammoniaco-magnesian phosphate. Whether the uræmic symptoms be due to urea or to carbonate of ammonia, it is unnecessary here to discuss (see p. 12). It is probable, however, that in uræmia, from diseases of the kidney, and in the typhoid state generally, the urea which is retained in the blood, is partly decomposed, and eliminated from the system in the form of a volatile salt of ammonia. This connection between the typhoid state and the presence of urea, carbonate of ammonia, or other products of disintegration of tissue in the blood, is a subject, which requires and deserves further investigation. The establishment of such a connection would simplify greatly our knowledge of disease.

It has already been shown, that convulsions occurring in typhus, have a uræmic origin, and that albuminuria is not uncommon in the typhoid state, even when there are no convulsions. In two cases also, where the typhoid state was well developed for some days before death, I have found urea in the blood. The following are the notes of one of these cases.

Case XIII.

Typhus proving fatal on the sixteenth day. Death preceded, for two days, by Stupor and Coma. Blood fluid, and containing Urea.

George M——, aged 69, admitted into the London Fever Hospital, July 21st, 1862. Had been an inmate of a workhouse, where his illness commenced with rigors, headache, and general pains, seven days before admission.

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[y] Frerichs, *Klinik der Leberkrankheiten*, Syd. Soc. Transl., i. 183.

[z] *Report on Yellow Fever, B. & F. Med. Chir. Rev.*, 1856, vol. xvii.

On admission, his chief complaints were headache and restless nights. He was a little confused, but answered correctly. Tongue moist and furred. Bowels opened by medicine. Typhus-rash well out. Pulse 72, and regular.

He was ordered a mixture containing sulphuric ether, sulphuric acid, and quinine; also 4 ounces of wine, beef-tea, and milk.

July 25th (twelfth day). Headache almost gone, and sleeps better; but is more prostrate, and tongue is dry along the centre.

Brandy was substituted for the wine.

July 27th (fourteenth day). Lies on his back, and is much more prostrate. Is more stupid and confused; but understands when spoken to. Tongue dry and brown. Pulse 90, and feeble. Water not passed in bed.

The brandy was increased to 10 ounces.

The same evening, he became very drowsy, and on the following day, he was quite unconscious; pupils contracted. Pulse 90, and feeble; skin dry; temperature in axilla, not exceeding 99·75° Fahr.

The patient was enveloped in a hot wet blanket, and then covered with dry blankets for three hours, while, at the same time, brandy was given freely. No improvement, however, took place; and on the following (sixteenth) day, the patient was much worse; pulse scarcely perceptible; surface livid and cold, and covered with perspiration; complete unconsciousness; contracted pupils, and floccitatio.

Death occurred at 5.30 p.m.

Autopsy, 22 hours after death.

With the exception of old adhesions of the pleuræ, moderate hypostatic congestion of the lungs, and slight hyperæmia of the liver and kidneys, the internal organs were healthy. There was no trace of disease in the intestines.

The blood, contained in the heart and great vessels, was perfectly fluid and black. Three ounces of the blood, when treated in the manner described under Case X., yielded crystalline scales of nitrate of urea.

5. *Stage of Remission or Crisis.* By crisis of a disease, is understood a sudden change to recovery, usually accompanied by some increased secretion. There are few acute diseases, in which a more rapid transition from unfavourable, to favourable symptoms, occurs, than in typhus, or in which the appetite returns so readily, and may be gratified with so little impunity. This has been a matter of constant observation by those, who have had an opportunity of closely watching the disease. Hildenbrand stated that the disease abated 'd'une manière très prompte.'[a] In 1840, Dr. Stewart wrote thus: 'All that I insist upon is, the frequent, I may

[a] Hildenbrand, 1811, p. 77.

'say the common, occurrence of a perceptible crisis, or what is 'vulgarly termed, a *turn* in typhus. I think I may appeal to the 'experience of every physician, and more especially of every 'resident clerk in the Fever hospital, whether they have not often 'been struck at seeing, during their morning visits, the glassy 'eye, the haggard features, the low muttering delirium, the stupor 'approaching to coma, the tremor, the subsultus, the carphology, 'the rapid, thready, tremulous, and intermittent pulse of the 'previous evening, the formidable array of symptoms in short, 'which seemed to indicate a speedy and fatal termination, ex-'changed for the clear eye, the intelligent countenance, the steady 'hand, the comparatively slow and firm pulse, and the returning 'appetite of approaching convalescence. To such cases as these, 'we might almost apply the Scripture phrase: 'At such an *hour* 'the fever left him.' In the great majority of cases we can point 'with precision to the *day*, on which amendment took place.'[b] 'La fièvre;' says Jacquot, 'tombe souvent avec une rapidité 'étonnante.'[c] Barrallier, the latest systematic writer on typhus, observes: 'Cette période (de remission) survient presque brus-'quement.'[d]

Improvement is sometimes ushered in by sleep. The patient, who for days has been delirious, and more or less unconscious, falls into a sound and quiet sleep, and awakes refreshed, rational, and quite another man. Careful researches are still required on the connection between the so-called critical discharges, and the resolution of the febrile symptoms. There is no doubt that amendment is often attended by moderate perspiration, and, in other cases, by diarrhœa, or a copious deposit of lithates in the urine. On the other hand, the urine may deposit lithates at any stage of typhus, which are often wanting at the time of crisis, while both diarrhœa and sweating may occur either naturally, or as the result of treatment, without bringing about any favourable change. Moreover, according to Traube's researches,[e] these evacuations, when they occur, are 'after-critical,' rather than critical, being always preceded by a considerable fall in the pulse and temperature; if this be so, they seem to be the result, rather than the cause, of the cessation of the fever. Dr. Todd was of opinion, that death often resulted from the very effort of nature to relieve the system, or from an excess of the critical discharges, and certainly profuse perspiration is rarely observed in typhus, except before a fatal event.

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[b] STEWART, 1840, p. 305. [c] JACQUOT, 1858, p. 148. [d] BARRALLIER, 1861, p. 72.
[e] TRAUBE 1853. [f] TODD, 1860, p. 175.

6. *Convalescence.* No sooner has amendment commenced, than convalescence advances rapidly. The tongue becomes clean and moist, the appetite is ravenous, and the bodily powers daily improve. Unless the patient has been in a weak state, prior to the attack, or convalescence be retarded by complications, three or four weeks usually suffice to restore perfect health and strength. By this time, indeed, it is not uncommon for the convalescent from typhus, to boast of an unwonted amount of freshness and bodily vigour. Falling off of the hair is the only inconvenience, which the patient usually experiences. It is rare, indeed, for typhus to lay the foundation of any serious organic disease.

Duration.

It is very important, in reference to prognosis and treatment, to be able to fix the duration of typhus. The mean duration is about fourteen days; it varies according to the strength of the poison, and the constitution and circumstances of the individual attacked; but in uncomplicated cases, it rarely, if ever, exceeds twenty-one days. Sometimes, it appears to exceed this limit, owing to the presence of some local complication; but there can be no greater mistake, than to confound the duration of the primary fever with the length of the illness. In 1856 and 1857, I ascertained the duration of the fever, in 53 uncomplicated cases which recovered, as follows:

In 1	case	convalescence	commenced on	8th day.
„ 2	cases	„	„	9th „
„ 3	„	„	„	11th „
„ 2	„	„	„	12th „
„ 10	„	„	„	13th „
„ 14	„	„	„	14th „
„ 9	„	„	„	15th „
„ 7	„	„	„	16th „
„ 2	„	„	„	17th „
„ 1	„	„	„	19th „
„ 2	„	„	„	20th „
53				

Thus, in nearly one-half of the total number, convalescence commenced on the thirteenth or fourteenth day, and in 40 cases, or more than three-fourths, on the thirteenth to the sixteenth day inclusive. The mean duration of the 53 cases was 14·13 days.

Again, of 11 fatal cases, death occurred on the eleventh day, in 2; on the thirteenth, in 1; on the fourteenth, in 1; on the fifteenth, in 2; on the sixteenth, in 1; on the seventeenth, in 1; on the eighteenth, in 1; on the twentieth, in 1; and on the twenty-sixth,

in 1. In the last case, however, the fatal event was due to secondary phlebitis. The mean duration of the ten first cases was 15 days.

In all of the 64 cases, the commencement of the disease was well-marked.

Little dependence can be placed on the statements, made by many writers, as to the duration of typhus, inasmuch as it has been shortened, on the one hand, by the admixture of cases of Relapsing Fever and Febricula, and lengthened, on the other, by the admixture of cases of Pythogenic fever, and by including local complications with the primary fever. The following results are free from such objections. In the early part of this century, Hildenbrand ascertained that the crisis ordinarily occurred, on the fourteenth day.[g] Of 55 cases noted by Henderson, at Edinburgh, in 1838-39, in which there was marked typhus-eruption, the mean duration was $13\frac{2}{3}$ days.[h] The average date, at which death occurred in 143 cases, was calculated by Dr. John Reid, at between the twelfth and thirteenth day.[i] In the Edinburgh epidemic of 1847-8, I remember that it was a common observation among the physicians and nurses, that the fever 'took a turn' on the fourteenth day. In 1849, Dr. Jenner fixed the duration of typhus at between fourteen and twenty-one days, and maintained that uncomplicated cases were never prolonged beyond the latter date. Of 18 fatal cases, in which he was able to ascertain the date of commencement of the attack, the average day of death was the 14·27th, one patient dying on the tenth day, and another, not until the twentieth. At Toulon, in 1855, Barrallier found that of 698 cases which recovered, convalescence commenced between the tenth and the twenty-second day, in all but 74, in which complications were present, and that of 436 fatal cases, death occurred, in the first week in 1, at the commencement of the second week in 44, at the end of the second week in 270, during the third week in 84, and at a later date, as the result of complications, in 37.[k] Lastly, the mean duration of 63 cases, observed by Godélier, in the hospital of Val de Grace, was between fourteen and fifteen days.[l]

But, although the duration of typhus is usually about fourteen days, and never exceeds three weeks, not a few cases run a much shorter course. Many cases are on record, where the disease has terminated fatally on the second or third day, or even after a few hours. Such were the cases of *Typhus Siderans*, or *Blasting Typhus*,

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[g] Hildenbrand, 1811, p. 78. [h] Henderson, 1839. [i] Reid, 1840.
[k] Barrallier, 1861, pp. 257, 368. [l] Godélier, 1856, p. 893.

which devastated the garrisons of Saragossa, Torgau, Wilna, and Mayence, during the wars of the first Napoleon.[m] Similar cases were observed in Ireland during the epidemic of 1847-8,[n] and among the French troops in the Crimea, in 1856.[o] From the testimony of several observers, both French and Russian, it appears, that the mean duration of typhus, in the Crimea, was only between twelve and thirteen days, in 1855, and between ten and eleven days, in 1856.[p] During the present epidemic (1862), in London, I have met with several instances, where death occurred as early as the eighth day.

Barrallier[q] has endeavoured to show that those epidemics are always most mortal, which have been characterized by the shortest duration of the disease; but although the statement is, to some extent, true, cases of short duration are occasionally mild.[r] In my own experience, mild cases of typhus (with the eruption) have sometimes terminated on the twelfth, or tenth, or even as early as the eighth day (see Case XIV.) It is probable, moreover, that the so-called Febricula, where the fever lasts only two or three days, and is not attended by rash, occasionally results from a small dose of the typhus-poison; at all events, cases answering to this description sometimes occur in the same family, and at the same time, as true typhus. According to Dr. W. T. Gairdner, typhus has of late years, not only become rare, and assumed a mild character, at Edinburgh, but its course has been shortened, so as rarely to exceed twelve days.[s] No such change has occurred in London: cases of short duration were as common in 1856 as they are now.

My observation lends no support to the applicability to typhus, of Galen's doctrine of critical days, which has been recently revived by Dr. L. Traube of Berlin.[t] According to this doctrine, the disease should terminate on one of the odd days, the seventh, ninth, eleventh, thirteenth, fifteenth, etc., and not on the intermediate even days. Still Traube's investigations (although it is doubtful if they refer to cases of true typhus,) are deserving of attention; and it is to be observed that by the term *day*, Traube implies, not a period of twenty-four hours, commencing at midnight, but, according to the example of Galen, *a day of the disease*, commencing with its first symptoms.

The two following cases are examples of typhus, of short duration.

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[m] Gaultier de Claubry, 1838; Ozanam, 1835, iii. 202.
[n] See *Irish Report*, 1848, viii. 92; also, Graves, 1848, i. 240.
[o] Jacquot, 1858, p. 140; Barrallier, 1861, p. 101. [p] Jacquot, 1858, p. 136.
[q] Barrallier, 1861, p. 102. [r] W. T. Gairdner, 1862, p. 165.
[s] Ib. [t] Traube, 1853.

Case XIV.

Typhus, with Convalescence commencing on eighth day.

Mary G——, aged 47, admitted into the London Fever Hospital, July 28th, 1857. On the 24th, she had been quite well, but on the 25th she was seized with shivering, headache, general pains, and nausea.

July 29th (fifth day). Pulse 84, and feeble; much headache; expression heavy, and is a little confused, but answers correctly. Skin warm and dry, with a well-marked typhus rash on the chest and abdomen. Tongue dry and brown. Some cough, with frothy expectoration, and sibilant and sonorous râles over chest.

Was ordered beef-tea and milk, 6 ounces of wine, a mixture containing ammonia and squill, and sinapisms to the chest.

Continued much in the same state until the morning of August 1st, (eighth day), when she felt and looked much better; pulse 72, eruption almost gone; tongue clean and moist, appetite good, and cough much relieved. From this date, she improved daily.

Case XV.

Typhus, fatal on the eighth day.

William W——, aged 30, admitted into the London Fever Hospital, April 9th, 1862. Was well on the 4th, but on the 5th was seized with shivering and headache, and took to bed at once.

On admission, pulse 100, and feeble; severe headache; tongue dry along the centre; bowels confined. No eruption.

Was ordered castor oil, a mixture containing nitre and nitro-hydrochloric acid, 6 ounces of wine, beef-tea, and milk.

On the following day (April 10), the typhus-eruption began to appear; and on the 12th, it was noted as copious. On the 11th and 12th, the patient had a good deal of delirium, and became very weak. On the 11th, he was ordered 4 ounces of brandy, and on the 12th, 8 ounces.

April 13th (eighth day). Much worse. Pulse almost imperceptible; skin cold, and face livid; eruption darker; copious perspiration. Scarcely conscious; pupils contracted; much low delirium, and occasional subsultus. Motions and urine passed involuntarily.

The head was shaved, and a blister applied to the vertex, while half an ounce of brandy was given every hour.

Death occurred at $10\frac{1}{2}$ p.m.

Relapses.

True relapses are extremely rare in typhus. I have never met with a case, in which, after complete convalescence and disappearance of the eruption, a relapse of febrile symptoms has been marked by a return of the eruption, or could not be traced to some local complication. 'I have never,' says Dr. Stewart, 'among 'thousands of cases seen a single case of relapse, in the proper sense

'of the term, after the symptoms had begun to decline.'[u] A similar remark is made by Dr. Jenner, and by most writers. According to Barrallier, relapses occurred within a few weeks of the first attack in 10 of 1302 cases, observed by him at Toulon; but no mention is made as to the presence of eruption, or the absence of complications, in both attacks.[x] Out of nearly 5,000 cases of typhus reported at the London Fever Hospital, during the last fourteen years, the following is the only instance of an attack of typhus, followed by a true relapse. The patient was under the care of Dr. Tweedie, and the case was reported by Dr. Buchanan, at that time, the resident medical officer.

Case XVI.

Typhus, lasting for two weeks; after a week's interval, a Relapse with a Recurrence of the Eruption, lasting upwards of a fortnight.

Ann B., aged 42, a nurse in the hospital, was admitted as a patient on October 28th, 1855, having suffered for five or six days, from great headache, and other symptoms of typhus. The night before admission, she had been delirious.

On admission, the pulse was 120; the skin was hot and dry, with a distinct typhus-rash. Tongue furred; bowels confined; much vertigo. The chief symptoms, after admission, were, sleeplessness, and occasional delirium. On November 3rd, the patient was much better; the pulse was only 70; but the rash was still distinct. On November 5th, all the cerebral symptoms had disappeared, and there were only faint remains of the rash.

After this, she continued to improve, and was walking about the house, when, on November 16th, after ailing for a few days, she became so ill, as to take to bed again. The typhus-rash re-appeared very copiously on that day. Tongue brown and dry; appetite gone; occasional delirium; pulse 120. On November 19th, pulse 120; tongue still dry and brown; great thirst; frequent delirium; urine passed in bed; and prostration immensely greater than in former attack. No cough. On November 24th, very restless and delirious at night; face flushed; pulse 120, and very weak. Rash still very freely out.

After this date, no report was made until December 10th, when the patient was stated to be convalescent, but to be suffering from extensive ulcers of the legs, which had followed the application of mustard poultices, for the purpose of rousing her from a state of stupor.

Section VIII.—Complications and Sequelæ of Typhus.

Many cases of typhus, during their course, present complications, which constitute no essential part of the primary disease.

[u] Stewart, 1840, p. 300. [x] Barrallier, 1861, pp. 262, 371.

This remark applies, with especial force, to the cases which terminate fatally, death being in many, if not most, instances, due to complications. Most complications commence before the cessation of the primary fever, in calculating the duration of which, it must be borne in mind, that, after its cessation, the illness may be prolonged in this way, to an indefinite length. After convalescence is fairly established, in uncomplicated cases, it is rarely interrupted by the occurrence of sequelæ.

The frequency of different complications varies, at different times and places. In some epidemics, scurvy is a common complication, in others, dysentery; while, as a rule, both are rarely observed. Parotid swellings, erysipelas, pyæmia and local gangrene are sometimes common complications; at other times, they are rare. Of 43 cases, examined after death in the Edinburgh Royal Infirmary, between April, 1838, and September, 1839, true pneumonia was found in only one instance;[y] whereas it existed in 11 out of 88 cases, examined between September, 1839, and September, 1841;[z] and during the next year (1841-2), out of 27 cases, there were two examples of pneumonia.[a]

a. Diseases of the Respiratory Organs.

Diseases of the respiratory organs are the most common complications of typhus, in this country. Their advent is often most insidious; for the ordinary symptoms, cough and expectoration, may be slight or absent, and the patient is unable to complain of pain. It often happens, that rapid breathing and lividity of the face, are the first indications of extensive disease of the lungs. Acceleration of the breathing, however, is not alone sufficient to indicate disease of the lungs; for, in grave cases, it is usually a symptom of the primary fever, and sometimes it is extreme, independently of any pulmonary disease, as in the so-called 'nervous respiration' (see p. 137). Again, although the dyspnœa arising from pulmonary disease is distinguished by the presence of lividity of the face and hands, owing to non-aeration of the blood, this appearance is only observed, when the local complication has attained to a great, and too often, to an irremediable extent. Hence, in every case, where there is the slightest doubt, the chest should be examined daily, or even oftener, by auscultation and percussion. For this purpose, the patient's strength will rarely enable him to sit up, but all the necessary information may be obtained by turning him on his side.

1. *Bronchitis* is perhaps the most common of all the complications of typhus. In some epidemics, it is present to a greater or less

[y] Reid, 1840. [z] Ib. 1842. [a] Peacock, 1843.

extent in most cases. So much is this the case, that in Ireland it has been the custom to speak of 'Catarrhal Typhus,'[b] while Rokitansky, and other German pathologists, believing in the identity of typhus and pythogenic fever, but drawing their knowledge of the former, chiefly from Irish sources, think that typhus merely differs from pythogenic fever, in the 'typhus matter' being localised in the lungs, instead of in the intestines.[c]

Bronchitis may usher in, or come on, at any period of, typhus, and may persist after the primary fever has ceased. All cases where it is present must be carefully watched. So long as the evidence of pulmonary disease is confined to occasional cough, and a few sibilant râles over the chest, there is no immediate danger; but as the general prostration increases, the pulmonary disease is very apt to extend suddenly and insidiously, and to be associated with more or less hypostatic consolidation. Moreover, owing to the patient's inability to cough, coupled with the impaired nutrition and paralysis of the muscular fibres of the bronchi, there is a remarkable tendency for the bronchial secretion to accumulate in the tubes, so as to threaten asphyxia.

2. *Hypostatic Consolidation*, exists, to a greater or less extent, in all severe cases of typhus. It usually commences at an advanced stage of the disease (eleventh to fourteenth day), when the general powers are weakest, but it may come on as early as the seventh day. Owing to the paralysed state of the pneumogastric nerves[d] interfering with the respiratory functions, and the diminished power of the heart, passive congestion takes place in the most dependent parts of the lungs, while at the same time serum is effused into the pulmonary tissue, and there is increased secretion from the lining membrane of the bronchi. Pulmonary hypostasis, in fact, is always accompanied by more or less bronchial catarrh, and it is this combination which is the most common cause of death in British typhus. This morbid condition, often mistaken for pneumonia, may come on suddenly, and rapidly extend to a fatal termination, in patients who have passed through the greater part of the attack, without any very unfavourable symptom. A patient, in this state, may have little or no cough or expectoration. Indeed, the absence of cough, betraying, as it does, the utter inability of the patient to rid the bronchi of the gradually increasing secretion, is an unfavourable indication. When there is expectoration,

[b] Lyons, 1861, p. 162. [c] Rokitansky, *Path. Anat.* Syd. Soc. Ed. ii. 74; iv. 24.
[d] Dr. John Reid showed that division of the pneumogastric nerves, in animals, produced appearances in the lungs, similar to the hypostatic consolidation, so common after death from typhus. (*Anat. and Path. Res.* pp. 199, 205).

it is tenacious and frothy, and often mixed with streaks or small masses of florid blood. The respirations are accelerated to 40, or even to 60, but are laboured and imperfect; the pulse is correspondingly quickened, weak, and often irregular; but occasionally, towards the end, both pulse and respiration are abnormally slow, the face and extremities are livid, the surface is cold and often clammy, and the patient is in a state of stupor passing into coma. On percussion of the chest, there is dulness, which at the first is confined to the most dependent part of either lung; as a rule, it is most marked, not at the bases, but about the middle, or a little lower. Gradually the dulness extends; sometimes it is more extensive over one lung than the other, but it is always most decided at the back part. On auscultation, mucous and subcrepitant râles are audible over the entire lungs,—over the resonant parts as well as the dull, while over the latter, the breathing is either feeble, absent, or bronchial. In one case, I have known death occur rather suddenly, as early as the seventh day, from non-granular consolidation of the lungs.

3. *Pneumonia.* True pneumonia, with exudation of solid lymph into, and between, the air-cells, is comparatively rare in typhus. The majority of the cases of so-called pneumonia, are examples of hypostatic consolidation with bronchial catarrh, as above described. When true pneumonia does occur, it is sometimes developed with great rapidity, the whole of one lung becoming solidified in a few hours. It is not always possible to distinguish it during life from pulmonary hypostasis; and, in fact, the lesions may exist together. If the dulness be limited to the base or apex of one lung, and if the sputa be rusty, it is probably true pneumonia that we have to deal with. According to Dr. Lyons,[e] pneumonia in typhus first implicates those parts of the lungs, which usually escape in ordinary pneumonia, as it attacks the upper and anterior parts, in preference to the lower and posterior. My experience does not confirm this observation. In the course of practice, I have met with fully a dozen cases of pneumonia, irrespective of typhus, commencing at the apices, and I have invariably noticed that the symptoms were of a remarkably low typhoid character; but I have never chanced to meet true pneumonia in this locality, as a complication of typhus. As a rule, the consolidation has been in some part of the lower lobe, and often it has been lobular. In several cases of typhus, however, I have known consolidation of the apices of the lungs produced by œdema.

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[e] Lyons, 1861, p. 171.

4. *Gangrene of the Lung.* Now and then, the pneumonia of typhus terminates in gangrene, which is recognised without difficulty, by the peculiar and horrible odour emitted with the breath, the sudden aggravation of the symptoms, and the pinched ghastly expression of countenance, together with the local signs of pneumonia. Two cases of this nature are recorded by Dr. Jenner,[f] and several have come under my own notice. In one or two instances, I have noticed, that the pulmonary gangrene was secondary to extensive bed-sores over the sacrum. Gangrene of the lung is chiefly observed in patients, who have been starving for many weeks prior to the attack of typhus.

5. *Pleurisy* is not a common complication of typhus. When it occurs, its advent is latent. No sharp pain is complained of; and, as a rule, the affection is not discovered until the effusion is so considerable, as to embarrass the breathing. The effusion is almost always fluid, and consequently friction is rarely to be heard.

6. *Tubercle* is occasionally deposited in the lungs, as a complication, or sequela, of typhus, although different opinions have been expressed on the point. Dr. Christison states, as the result of his extensive experience in fever, that consumption is a very rare result of true typhus, and that its origin in typhus as a predisposing cause is very problematical, in any instance.[g] Stokes[h] and Huss[i] on the other hand, insist much on typhus predisposing to pulmonary tubercle, although it may be doubted, if many of the cases, from which their conclusions are drawn, were not examples of pythogenic fever, which is more frequently followed by tubercle. Jenner, however, records an instance where a rapid fresh deposition of tubercle in the lungs occurred during typhus, in a phthisical child[k]; and Dr. Stewart informs me that he has met with not a few cases, in which pulmonary phthisis has commenced during, or immediately after, an attack of typhus. I have observed three or four examples of the same nature. In these cases, there were all the signs, during the fever, of bronchitis or pneumonia, which persisted after its cessation, when rapid emaciation, profuse sweating, and purulent expectoration, took the place of convalescence. Still, according to my experience, tubercle is not a common sequela of typhus, and, in most cases where it occurs, there has been a prior phthisical history.

7. *Laryngitis* is an occasional complication of typhus. It consists

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[f] JENNER, 1849 (2), and 1850, xx. 456.
[g] CHRISTISON, 1840.
[h] STOKES, 1854.
[i] HUSS, 1855, p. 216.
[k] JENNER, 1850, xx. 457.

in an erysipelatous condition of the mucous membrane of the larynx, often associated with a similar condition of the pharynx, or with erysipelas of the face. It may be very insidious at its commencement. Great prostration and œdema glottidis are the dangers to be apprehended from it; but as a rule, it only causes slight hoarseness. In most cases, it is preceded by a similar condition of the pharynx.

b. Diseases of the Blood and Organs of Circulation.

1. *Phlegmasia dolens* or *White Leg.* During convalescence, an affection of one of the lower extremities is occasionally developed, which resembles closely what is known, as the phlegmasia dolens of puerperal women. Stokes states, that if, in convalescence from fever, the pulse continue very rapid, without any local cause, either in the chest or the abdomen, this complication may be anticipated. It always appears after the cessation of the primary fever, which has often been remarkably mild. According to Dr. Tweedie, it was formerly most common in cases where bleeding had been practised to a large amount, while it is most apt to occur in the parturient female, when delivery has been followed by extensive uterine hæmorrhage. The altered principles of treatment in fever may possibly account for the comparative rarity of the complication, at the present time. Out of nearly 700 cases of typhus, which have come under my care in the present epidemic (1862), it has not occurred in a single instance.

The term phlegmasia dolens is not always strictly accurate, for the affection is not always painful. Sometimes, there is so little pain, that the discovery of the local disease is entirely accidental; but, in most cases, there is considerable pain, and also tenderness, in the iliac fossa, and along the femoral vein; and occasionally shooting pains in the extremity are complained of, for some days before any swelling appears. Many of the patients are seized, during the night, with severe pains in the calf; and in the morning, the whole, or part, of the corresponding lower extremity is tense and swollen. The swelling is usually of a firm brawny character, and sometimes it is enormous. The skin of the entire body, but particularly that of the affected limb, is pallid. The femoral vein can sometimes be felt like a hard cord; and, in some instances, a corded condition of the superficial veins is observed. There is seldom any great constitutional disturbance; there is no nausea, vomiting, or jaundice,[1] and most cases terminate favourably, the

[1] In one case I have noticed slight jaundice. (See Case xvii.)

swelling gradually subsiding, but often leaving a hard cord in the situation of the femoral vein. Occasionally, however, the swelling lasts for several weeks; and then, according to Corrigan, it is usually succeeded by a varicose condition of the superficial veins, which may last for life. Usually, it is the left leg that suffers, and there is but one attack; but now and then, one leg recovers, and, after a week or ten days, the other is attacked; while even a third seizure may occur, in the limb first affected.

Examples of this affection were observed at Edinburgh and Dublin, during the epidemic of 1817-19,[m] but Dr. Tweedie was the first to direct attention particularly to the subject, in 1828.[n] Although the dependence of puerperal phlegmasia dolens on phlebitis had been rendered probable five years before, by the researches of Dr. David Davies,[o] Bouillaud,[p] Velpeau, and afterwards by Dr. Robert Lee,[q] Dr. Tweedie made no mention of the condition of the veins in the corresponding affection after fever, which he attributed to 'inflammation of the cellular tissues 'of the limbs.' Most modern pathologists, however, have ascribed post-febrile, as well as post-partum, white leg, to plastic phlebitis, or the formation of a thrombus in the iliac or femoral vein. This opinion has been chiefly based on the hard cord-like condition of the veins, often felt during life; there have been few opportunities of examining their condition after death; for, as a rule, the patient recovers. The phlebitic theory has been supported by the statement, that extensive bed-sores account for the origin of the post-febrile phlebitis,[r] in the same way as the raw surface of the uterus is believed to be the first step, in the pathology of that which follows parturition. But Dr. Stewart observed several cases at Glasgow, in 1838, where there were no bed-sores,[s] and more than once, I have made a similar observation.

Two years after the appearance of Dr. Tweedie's memoir, Drs. Graves and Stokes[t] published some observations on 'Painful Swellings of the Lower Extremities,' in which they maintained the pathological identity of phlegmasia dolens occurring after delivery, and the painful swelling which succeeded fever; but they insisted that phlebitis 'could not in justice be considered as the 'cause of the disease,' as it was often absent. They agreed with Dr. Tweedie, that the disease consisted primarily, in inflammation of the subcutaneous cellular tissue of the limb. Similar opinions

[m] Barker and Cheyne, 1821, i. pp. 467, 490; Christison, 1840, p. 145.
[n] Tweedie, 1828. [o] *Med. Chir. Trans*, May, 1823, vol. xii. p. 419.
[p] *Archiv. Gén. de Méd.* Jan. 1823, sér. 1, tom. ii, p. 192.
[q] *Med. Chir. Trans.* 1828. [r] J. R. Bennett, 1857. [s] Stewart, 1857.
[t] Graves and Stokes, 1830.

were afterwards expressed by Dr. Graves in his 'Clinical Lectures,'[u] where it is stated, that phlebitis is not the first link in the morbid chain, and that it is merely a consequence of some unknown cause, which determines the inflammation of the other tissues. More recently, the same views have been urged by Dr. Mackenzie,[x] according to whom, phlebitis is not essential to phlegmasia dolens, but both are the result of some morbid condition of the blood. These views are confirmed by the only case of phlegmasia dolens after fever, that I have had an opportunity of dissecting. Here, the femoral and iliac veins of the affected limb were perfectly normal, and free from coagulum.

CASE XVII.

Typhus Fever, followed by Phlegmasia Dolens, Jaundice and Death. Autopsy. Fatty Heart. Acute Atrophy of liver. No clot in femoral vein.

Rosetta J. ——, æt. 42, admitted into the London Fever Hospital February 24th, 1857.

This patient had been ill for about eight or nine days, before admission; and after she came under observation in the hospital, her most prominent symptoms were: pulse 120, great prostration; great restlessness and much low muttering delirium; involuntary stools and urine; well-marked typhus-rash; dry, brown tongue, and constipated bowels. The treatment consisted in wine, carbonate of ammonia, and castor oil to keep the bowels open.

About five or six days after admission, an improvement took place in the symptoms; and, by the 6th of March, she had regained her strength, to a considerable degree, her appetite was good, and pulse 80.

On March 9th, which was about the 23rd day from the first commencement of the fever, and the 6th of convalescence, the patient felt ill again. Pulse 120, and small. Complained much of shooting pains in the left leg. Skin hot and dry. Some flushing of face. Tongue moist and very red.

The next day there was considerable swelling, and some tenderness of the left leg and thigh. The heart's action was heaving and tumultuous, but there was no bruit. Breathing short and rapid; no cerebral symptoms.

Blister over heart. Wine ℥vi. Saline efferv. mixture with Tinct. Hyoscy. ʒss. every four hours. Left leg to be fomented and kept elevated.

No improvement took place, but at 4 a.m. of March 12th (the fourth day from first complaint of pains in the limbs), the patient felt cold and chilly. There was a great increase of the prostration, and the pulse was imperceptible, although the heart's action continued tumultuous, as before. Breathing very rapid. The mental faculties were unimpaired. The skin and conjunctivæ were of a marked yellow tint, and the face livid. Profuse sweating. No tenderness over liver, nor obvious increase of hepatic dulness.

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[u] 2nd ed. 1848, i. 264. [x] *Med. Chir. Trans.* 1853, and LETTSOM. *Lect.* 1862.

Brandy and wine were freely administered, but the patient gradually sank, and died towards evening.

Autopsy, on March 13th.

Cadaveric rigidity well marked. Distinct yellow tinge of skin on scalp, neck, and trunk. Thick layer of subcutaneous fat over the chest and abdomen.

Copious crop of sudamina over chest. Left leg swollen. Left ankle $8\frac{3}{4}$ inches in circumference, right $8\frac{1}{4}$; left calf 13 inches, right $11\frac{1}{2}$; left thigh 17 inches, right $14\frac{1}{2}$.

Cerebral membranes moderately congested, and separated readily from brain. Sub-arachnoid fluid, and fluid in ventricles, normal in amount, but of a decidedly yellow tint, especially the fluid in left lateral ventricle. Substance of brain tolerably firm; red points pretty numerous. Brain weighed 42 ounces. Left choroid plexus much yellower than right.

Half an ounce of serum in pericardial sac. Heart $8\frac{3}{4}$ ounces, valves normal; left cavities empty, and right almost empty. Walls of right ventricle very thin, and at apex composed almost entirely of fat. Substance of heart pale and soft, and, on microscopic examination, transverse striation indistinct, and fibres presented a granular aspect. Left femoral and iliac veins healthy, and contained no clot.

Each of lungs weighed 25 ounces; left, adherent throughout, and very emphysematous. Lower lobes of both lungs much congested. No consolidation,

Stomach and intestines healthy.

Liver 52 ounces. Capsule separated readily. Substance of organ very soft and friable, so that it broke down on removal. All trace of lobules had disappeared, the cut surface of the organ presenting a marrow-coloured pulpy appearance. On microscopic examination, many of the secreting cells could be seen loaded with oil; others appeared to be breaking up and disintegrating; and there was much free oil and granular matter. A small quantity of thick bile in gall-bladder.

Spleen 13 ounces, soft and pulpy.

Kidneys enlarged; left $7\frac{1}{2}$ ounces, right 7 ounces; capsules separated readily; outer surface smooth; substance pale and flabby; cortical substance pale and granular, and rather increased in amount; uriniferous tubes gorged with epithelium.

2. *Pyæmia, with purulent deposits in the joints,* is occasionally noticed in severe cases about the period of crisis, or more commonly, during convalescence. Fortunately, the complication is of rare occurrence, as it is invariably fatal, within two or three days. Drs. Stewart and Anderson,[z] however, met with it not unfrequently at Glasgow, in the epidemic of 1836-38. It is ushered in by severe rigors, which are followed by great prostration and

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[y] Stewart, 1857. [z] Anderson, 1840, p. 49 and 1861, p. 48.

præcordial anxiety, extremely rapid and feeble pulse, swelling, redness, and tenderness of the joints, together with all the ordinary symptoms of pyæmia. There is almost always more or less jaundice, and often profuse perspirations. Sometimes scarcely a joint escapes, and many, even of the smaller joints, are filled with pus. After death, the synovial membranes are much injected, and bathed with pus, but are free from ulceration; and purulent deposits are rarely found in the internal organs.

Huss[a] and others have referred such cases, as now described, to suppurative phlebitis, which has been thought to originate in the absorption of pus into the veins from bed-sores. Dr. Stewart, however, states, that in some of his cases there were no bed-sores. Moreover, the opinion is gradually gaining ground, that pyæmia is always independent of the absorption of pus from a suppurating surface external to the blood-vessels, and that it originates in a putrid condition of the blood itself.[b] Like surgical fever, it is probably sometimes due to foul air, from over-crowding and defective ventilation. I have never known of a case at the London Fever Hospital.

3. *Scurvy* is another blood-disease which occasionally complicates typhus, but it is only observed in some epidemics. It contributed to increase the number of deaths from typhus, in the French army in the Crimea, and in the epidemic of 1847-8 at Edinburgh and elsewhere. Purpura-spots and vibices are very common in typhus so complicated; and in the Crimea, hæmorrhages from the nose, stomach, intestines, lungs, and bladder, were often observed. Great anæmia and tendency to syncope are other characteristics of such cases.

4. *Diseases of the Heart.* Both pericarditis and endocarditis are extremely rare complications of typhus. In only one instance, have I known endocarditis developed, during an attack of typhus. The remarkable change in the muscular tissue of the heart, which accounts for the impairment of the impulse and first sound (see page 136), will be subsequently described. In cases which recover, the cardiac action usually resumes its normal character with convalescence, except that the pulse is often at first unusually slow. I have known it not exceed 40, and, in one instance, it was not more than 36. This anomaly, however, almost always disappears, within a week or ten days. In the following case, about which there are many other points of interest, there is some reason to believe that the action of the heart was permanently impaired by an attack

[a] Huss, 1855, p. 206.

[b] See discussion at London Medical Society, *Brit. Med. Journ.* Oct. 26, 1861.

of typhus. The patient was a medical friend, to whom I am indebted for the account of the case.

Case XVIII.

Typhus, followed by General Paralysis and Anasarca, and remarkably Slow Pulse.

A gentleman, now about 33 years of age, had always enjoyed excellent health till March, 1854, when he left England, as Staff-Assistant Surgeon, with the army of the East. His pulse had averaged from 60 to 70. In August, 1854, he suffered from a severe attack of remittent fever, which nearly proved fatal by hæmorrhage from the bowels. Refusing to be invalided to England, he joined the expedition to the Crimea, on the 4th of September, and shared the fatigue and privations of the army before Sebastopol, during the following winter. In the beginning of May, 1855, he caught typhus from a number of Spaniards, who were under his care. The attack was severe, being characterized by a copious rubeoloid rash, great and protracted delirium, involuntary stools, and inability to swallow. The treatment consisted in stimulants, and numerous blisters to the neck and behind the ears. On recovery from the primary fever, the blistered surfaces took on unhealthy action and sloughed; the patient remained very prostrate, and became greatly emaciated, while the legs were œdematous, and the abdomen was thought to contain fluid. It is uncertain, whether or not the urine contained albumen. On the application of sulphate of copper to the ulcerated surfaces, the profuse discharge suddenly ceased; but the day after, the patient had an epileptiform fit, which lasted about two minutes. For this he was purged and kept on low diet, while the dropsy was treated with squills and nitric ether. Under this treatment, he became much weaker, and lost flesh; his eye-sight became impaired; and for a fortnight, he was unable to read large type, or even to distinguish large objects plainly. About the same time, he began to complain of pricking sensations—first in the toes, and afterwards in the fingers—and inability to perform the finer duties of the fingers, such as buttoning his shirt. These pricking sensations gradually extended up the limbs, and were followed by numbness and loss of power, ending with complete paralysis of both motion and sensation, although the slightest handling of the calves of the legs excited the most exquisite pain. The tongue was also œdematous, and was the seat of pricking sensations; while the muscles of deglutition were paralysed, so that swallowing could only be performed slowly, and required the attention of the mind. The integuments of the abdomen were devoid of sensation, but there was no paralysis of the rectum or bladder. The mind was unimpaired.

In this state, the patient arrived in England in Aug. 1855, and was seen by Dr. Todd, who suggested good diet, and a cold splash-bath, night and morning. After the first bath, he acquired some motion in his hands, and, by the end of a month, he was able to stand; and in two weeks more, he

could walk about. During his recovery, tinnitus of the right ear came on, and continued with scarcely any intermission for two years; it was always increased by fatigue, and relieved by a glass of wine. With this exception, he progressed favourably; and, in about eight months from the date of his arrival in England, he was able to follow the duties of his profession; although ever since, he has suffered from a slight feeling of weakness, and from inability to bear much bodily fatigue.

One day in March, 1858, after seeing some hospital patients, he suddenly felt his heart working in a strange manner, thumping slowly; and, at the same time, he experienced a feeling of giddiness. The pulse was barely 40; its rate since the attack of fever in 1855 is uncertain, but it is believed to have been normal. There was no cause, such as over-exertion or fatigue, to account for the attack. Next morning, the pulse remained the same; and the patient suffered greatly from muscular fatigue, and dyspnœa on going up stairs, requiring him to halt every few steps, and also from cold extremities, and difficulty in maintaining the animal heat. He was confined to bed, and treated with alcoholic stimulants and large doses of quinine. About a fortnight from the commencement of this attack, after a strong purgation, the patient experienced a sudden feeling of warmth, and the heart began to beat at its proper rate.

He continued well until the following October, when one morning, while sitting in a friend's house, he suddenly felt his heart change its action, and, on rising, he felt giddy. From that time to the present date (June, 1862), the pulse has varied from 34 to 36, and once it was as low as 33; it is regular, and there is no abnormal sound with the heart; the number of pulsations is not affected by any form of excitement, but each beat is then made with greater force, and thus a sense of disagreeable thumping is produced. For this reason, he seldom drinks anything stronger than milk, tea, or toast and water. He is still unable to walk up steep ascents, or to bear much bodily exercise. In all other respects, his health is remarkably good; he is able to discharge the onerous duties of his profession, and can bear a large amount of mental fatigue.

c. Diseases of the Nervous System.

1. *Mental Imbecility and Mania.*—As a rule, the intellectual faculties are completely restored, after the first few days of convalescence; but occasionally they remain blunted, for some days or weeks, after the patient has regained sufficient strength to walk about. The mind does not recover as quickly as the body. The memory is defective, the patient takes a long time to answer questions, mistakes one person for another, fancies that he has seen friends who have not visited him, and is strange and childish in his manner. Mania has been described by several writers as an occasional sequela. I have repeatedly known patients noisy and delirious, and getting out of bed for no object, several days after

the pulse had fallen, the tongue become clean and moist, and the appetite restored: in two instances, I have met with actual mania, protracted over many weeks, and in one case, necessitating the patient's temporary confinement in a lunatic asylum. Roupell records the case of a female, who was maniacal for six months after an attack of typhus, and was confined in a lunatic asylum, but recovered after a miscarriage.[c] These sequelæ are chiefly observed in cases, where the fever has been characterized by great and protracted delirium, and where there is reason to believe, that the nutrition of the brain has been more than usually impaired. There is no evidence, that they depend on any organic change in the brain of an inflammatory nature, for they are attended by no fever or headache, and, as far as my experience and reading extend, the mental faculties are invariably restored at last.

2. *General Convulsions.*—(See page 160).

3. *Partial Palsy* is a remarkable, though not common, sequela of typhus. Incontinence of urine during convalescence is, perhaps, the simplest, and most common form; but at other times, the deltoid muscles, or the muscles of an entire limb, or even of the face, are paralysed, or the paralysis presents itself, in the form of paraplegia or hemiplegia. In a case already mentioned, there was complete paralysis of both upper and lower extremities. (Case XVIII). The paralysis of motion may be complete, but it is oftener incomplete. It is often preceded by severe pain, or pricking sensations, and is accompanied by slight numbness, or complete anæsthesia, or hyperæsthesia, of the affected part. As a rule, it wears off with the return of general health and strength, but I have known it persist for months or years, while occasionally, after power is restored, the affected limb remains atrophied for life. This paralysis has been referred to organic affections of the head and spine, the existence of which is doubtful, as the patients almost invariably recover, and the mind is unimpaired. Although the nervous system may be primarily at fault, the palsy is really due to an exaggeration of the muscular atrophy, which, to some extent, is always produced by typhus. The muscles, in extreme cases, are shrivelled, pale, and in a state of fatty degeneration. Some muscles in a limb may be more atrophied than the rest, and thus club-foot and other distortions may be produced. An instance, of this nature, has come under my notice. Barrallier met with two cases of temporary hemiplegia during convalescence, and similar cases are mentioned by Huss.[d]

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[c] Roupell, 1839, p. 176. [d] Huss, 1855, p. 225; Barrallier, 1861, p. 255.

4. *Muscular Pains.*—Aching pains, in different parts of the body, may occasion no small distress during convalescence. Their precise nature is obscure; but they seem to have their seat in the muscles, and they usually cease after a few days. Occasionally, the patient complains of severe pains in the feet and legs, which have almost a neuralgic character, and which ought always to excite attention, as they often precede phlegmasia dolens, gangrene of the feet, or paralysis.

d. Diseases of the Organs of Special Sense.

1. *Deafness,* which is so common a symptom during the fever (see page 170), now and then persists during convalescence. As a rule, it ceases in a few days, but, according to Huss, it is sometimes permanent.[e] Sometimes it is associated with buzzing sounds in the ears, which may be so constant and distressing, as to prevent sleep. These symptoms may be connected with otorrhœa, or with inflammation of the internal ear; but often nothing abnormal can be discovered in the ears. In every case, however, where deafness and ringing in the ears persist after the cessation of the fever, the ears ought to be examined. I have known intense headache and delirium occur during convalescence, and cease at once, on the appearance of discharge from the ear. Dr. G. A. Kennedy relates instances, where otorrhœa was preceded by profound coma, dilated insensible pupils, and involuntary stools.[f] Graves records an instance, where there was reason to believe, that inflammation of the outer ear spread to the membranes of the brain.[g]

2. *Amaurosis.* During convalescence from severe attacks, there is occasionally slight dimness of vision, which ceases after a few days.

e. Diseases of the Organs of Digestion.

1. *Pharyngitis.* Erysipelatous inflammation of the mucous lining of the pharynx is met with, in some cases of typhus. It may precede, accompany, or succeed, erysipelas of the face. It often gives rise to considerable difficulty in swallowing, fluids being retained for some time in the mouth, or rejected by the nostrils. The dangers to be apprehended from it are, interference with nutrition, and œdema glottidis.

2. *Diarrhœa.* As already stated, (page 142) constipation is the rule in typhus, but occasionally diarrhœa is present as a complica-

[e] Huss, 1855, p. 223. [f] G. A. Kennedy, 1838, p. 28. [g] Graves, 1848, i. 191.

tion. Diarrhœa, early in the disease, is usually due, to the administration of purgatives; when spontaneous, it more commonly appears about the period of crisis.

3. *Dysentery.* Relaxation of the bowels, in typhus, may be due to dysentery. This is not a very common complication in Britain; but in some of the Irish epidemics, and in the French army in the Crimea, typhus and dysentery prevailed together, and occasionally complicated one another (see page 114). When typhus is complicated with dysentery, there may be tympanitis, diarrhœa, abdominal pain, and hæmorrhage from the bowels, and if the rash be absent, it may be difficult to distinguish it from enteric fever.

4. *Jaundice* is a common symptom of relapsing fever; but in typhus, it is extremely rare. Dr. Jenner never met with an instance. Jaundice, however, is occasionally observed. Dr. Henderson refers to such cases;[h] two cases are recorded by Frerichs in his work on 'Diseases of the Liver,'[i] and two have come under my own notice; they are almost invariably fatal. No obstruction of the bile-ducts is found after death; but, as in pyæmia, yellow fever, cases of snake-bite, and other blood-poisonings, the jaundice is probably due to some abnormal condition of the blood. For the latest views on the pathology of these forms of jaundice, the reader is referred to the work on 'Diseases of the Liver' by Professor Frerichs, of Berlin.[k] In one of my cases (p. 189), the liver after death resembled the appearances, presented in Acute or Yellow Atrophy: it was not much reduced in size, but it was pale-yellow and extremely soft; it exhibited no trace of division into lobules, and, under the microscope, there was an immense amount of oily and granular matter, while the secreting cells appeared to be undergoing disintegration. Frerichs found leucine and tyrosine in the hepatic tissue of his cases; hitherto, these substances (see page 149) have been chiefly found in the liver, kidneys, and urine, in cases of acute atrophy of the liver.

Leucine and tyrosine were also present in the following case, in which they appeared to be substituted for urea in the urine.

CASE XIX.

Typhus complicated with Jaundice. Death by Coma. Leucine and Tyrosine, but scarcely any Urea, in Urine. Leucine and Tyrosine in Liver and Kidneys.

Robert R——, aged 33, admitted into London Fever Hospital, August 26th, 1862.

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[h] HENDERSON, 1843, p. 220.
[i] Syd. Soc. Transl. i. pp. 168, 170.
[k] Ib. pp. 157, 174.

On admission, was too confused to give any account of himself; pulse 120, feeble; tongue dry and brown along centre; skin warm and dry, with distinct typhus-rash, and a general yellowish tint. Was ordered beef-tea, milk, brandy (6 ounces), and a mixture containing sulphuric acid, sulphuric ether, and quinine.

The patient became weaker, and more unconscious. On the 28th, there was decided jaundice of the entire skin, and of the conjunctivæ; the brandy was increased to 8 ounces.

August 29th; pulse 120 and feeble; is scarcely conscious, and is inclined to be drowsy; pupils contracted. Decided jaundice of skin and conjunctivæ, and, at the same time, a well-marked petechial typhus-rash on chest and abdomen. Involuntary evacuations; tongue brown; motions light-coloured, but contain bile; no tenderness in hepatic region; urine of a bilious colour, but does not yield the reaction of bile-acids; clear; acid; throws down no deposit; and contains no albumen. Specific gravity, 1017. Six ounces of the urine were evaporated, and the residuum was found to contain abundance of globular masses of leucine, and needle-shaped crystals of tyrosine, and also crystals of triple phosphate. When nitric acid was added to a drop of the urine, after concentration to one-twelfth of its volume, only a few small crystals of nitrate of urea could be discovered with the microscope.

A blister was applied to the scalp; but the patient died comatose, at 3 p.m., on August 30th.

Autopsy, 20 hours after death.

Deep jaundiced tint of entire surface; heart and lungs healthy; blood, fluid and dark; spleen, 7 ounces, very soft; liver, 62 ounces, rather pale, and very friable, but lobules distinct. Hepatic tissue contained numerous globular crystalline masses of leucine and tyrosine; secreting cells loaded with oil and bile pigment; kidneys enlarged, each weighing upwards of 7 ounces; surface smooth; cortex hypertrophied, and containing crystalline bodies, similar to those found in the liver; uriniferous tubes gorged with epithelium; intestines healthy.

5. *Peritonitis* is almost unknown, as a complication of typhus. Dr. Jenner, however, has recorded the case of a girl aged 16, who died from acute idiopathic peritonitis, commencing suddenly on the fifth day of convalescence; the ileum and mesenteric glands were found perfectly healthy.[1] Another case of typhus lately proved fatal from peritonitis, at the Fever Hospital, under the care of Dr. Buchanan. Here, the peritonitis resulted from the bursting

[1] Jenner, 1850, xxii. 408.

of a softened embolic deposit in the spleen; the mitral valve was covered with soft vegetations; the ileum was healthy. In one case, I have known death result from tubercular peritonitis, shortly after an attack of typhus.

f. Diseases of the Urinary Organs.

1. *Disease of Kidneys.*—From what has already been stated (pages 149, 162), it is obvious, that there can be no more serious complication of typhus, than disease of the kidneys, whether this disease be of old date, or the result of the primary fever. The obstructed elimination of urea poisons the blood, and induces typhoid symptoms of an intense form, such as stupor, coma, and occasionally convulsions.

g. Complications referrible to the Organs of Generation.

1. *Profuse Menstruation.*—Copious discharges of bloody fluid from the vagina occasionally occur in the advanced stages of typhus, and may increase the prostration.

2. *Pregnancy.*—Pregnant females are not exempt from typhus, and women, in an advanced stage of pregnancy, may pass through the disease without miscarrying. When miscarriage does occur, it is not necessarily fatal, either to the mother or infant. I have known it happen in 4 cases of typhus; but in all, both mother and child did well. Wardell also says that, at Edinburgh, pregnant females, in typhus, had no disposition to miscarry.[m]

h. Diseases of the Integuments, &c.

1. *Erysipelas* is an occasional complication of typhus. It may appear as early as the fifth day; but, as a rule, it is not observed until the end of the second or the third week, and in many cases it does not appear until convalescence. It usually commences at one side of the nose, and spreads over the face and scalp, and is sometimes accompanied by a similar condition of the pharynx. Other parts of the body are not exempt. It may be attended by delirium, coma, and other head symptoms, and always adds greatly to the severity of the case. It often terminates by the formation of abscesses in the eyelids, or beneath the scalp. Some patients exhibit a remarkable liability to erysipelas, which, after disappearing, recurs repeatedly in the same place, or in other parts of

[m] Wardell, 1846.

the body. When many cases of erysipelas appear in rapid succession in a ward, they may often be traced to overcrowding or defective ventilation, or to some patient with foul and offensive bed-sores, in the same ward.

2. *Œdema.* Slight œdema of the feet and toes, arising from debility, is sometimes observed, when the patient begins to walk. It seldom lasts longer than a week. In rare cases, of which an example will be found at p. 191, there is general anasarca, which is probably connected with disease of the kidneys.

3. *Gangrene from Pressure.* Bedsores are not uncommon in cases which are protracted from other complications; but, in uncomplicated typhus, they are, according to my experience, rare. A similar observation has been made by Barrallier.[m] Their most common situation is over the sacrum, but they also form on any part of the body subjected to pressure, such as the trochanters, heels, occiput, ears, elbows, the lower angles of the scapulæ, and the spines of the last cervical and first dorsal vertebræ. These bed-sores commence as an erythematous patch, which, after a time, becomes hard and black in the centre. By and bye, a line of demarcation forms between this central dark part and the surrounding erythema; the central part becomes more and more detached, and at last separates as a slough, leaving a dirty excavated ulcer, which may extend by sloughing, ulceration, or burrowing, beneath the surrounding integuments. Bed-sores greatly protract the duration of the illness, and may themselves endanger life by the great amount of discharge, or by inducing other complications, such as gangrene of the lungs. (See page 185.)

4. *Spontaneous Gangrene.* Parts, free from pressure, are not exempt from gangrene, in typhus. Occasionally, gangrene commences in the toes and spreads upwards, involving all the tissues down to the very bones. At Edinburgh, in 1848, I saw a patient who lost both feet from this cause; the gangrene extended to some inches above the ankles, where a line of demarcation formed, and both legs were amputated below the knee. During the present year (1862), in London, I have had one patient who lost the toes of both feet, and a second, who lost the greater part of one foot, in a similar manner. It has been suggested, that the gangrene in such cases is due to plugging of the arterial trunks; but I am not aware, that it has been proved to be so, by *post-mortem* examination. The majority of the patients have been in a state of star-

[m] Barrallier, 1861, pp. 96, 220.

vation, for weeks prior to the attack of fever, and the gangrene appears to be the result of extreme feebleness of the circulation. It is usually preceded by severe shooting pains in the legs and feet, with coldness and lividity of the toes.

Sloughing or ulceration of both corneæ, I have met with in several instances, and similar cases are mentioned by Jenner[n] and Huss.[o] The affection appears to be partly due to the eyes being kept constantly open, from paralysis of the orbiculares muscles.

The nose,[p] penis, and scrotum, have likewise been observed to slough. Dr. Lyons records a remarkable case, where the whole of the integuments, over the anterior and superior part of the chest, sloughed; the patient, at the time of attack, was in the last stage of starvation.[q]

5. *Noma*, or *Cancrum Oris* is a very fatal form of gangrene, which attacks the mouth, tongue, and face. It is most common in children, and is met with in severe cases of measles and small-pox, and in some other diseases, as well as typhus. It usually commences about the end of the second week, in the form of gangrenous ulceration of the mucous lining of one cheek. The external integuments become enormously swollen, red, tense and painful. By and bye, a dark speck, like a spot of purpura, appears at about the centre of the external swelling, corresponding to the situation of the internal ulceration. This speck rapidly enlarges to the size of a half-crown piece, and becomes surrounded by a rim of ulceration, by means of which the central slough is gradually detached, disclosing the interior of the mouth. The corresponding side of the tongue is likewise, more or less, implicated. Three or four days are usually sufficient to put an end to life; death, indeed, may occur before any attempt at separation of the slough. This complication was well described, forty-three years ago, by Dr. Marshall Hall;[r] and an excellent coloured representation of the disease has been published by Dr. G. A. Kennedy.[s] In the Crimea, it was a not uncommon complication of typhus, and was always fatal. Its occurrence has been attributed to the abuse of mercury; but it is met with in cases, where mercury has never been administered.[t]

6. *Hospital Gangrene.* Wounds and ulcerated surfaces are very

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[n] JENNER, 1850. [o] HUSS, 1855, p. 229. [p] M'GRIGOR, 1809. [q] LYONS, 1861, p. 191.
[r] HALL, 1819. [s] G. A. KENNEDY, 1838.

[t] Some writers restrict the term 'Cancrum Oris' to gangrenous ulceration, commencing in the gums, and spreading to the lips and cheeks, but not producing sloughing of the entire thickness of the cheek; while they apply 'Noma' to the affection above described (See *Chelius' Surgery*, South's ed. i. 62).

apt, under the influence of the typhus-poison, to degenerate into hospital gangrene. South records an instance, where an ulcer of the leg, which had existed for eighteen months, assumed all the characters of spreading gangrene, on the patient being attacked with typhus.[u] Jacquot states, that, during the prevalence of typhus in the French hospitals in the East, wounds of every description were extremely prone to degenerate into hospital gangrene, and that it was impossible to apply blisters, without a similar risk.[x] Similar observations were made by Larrey;[y] others have been collected by Barrallier;[z] and the fact must be familiar to every physician, who has had much experience in typhus. It is not necessary, that the patient, with the wound or ulcer, should contract typhus himself; mere exposure to the typhus-poison, or to the conditions capable of generating it, is sufficient. Hospital gangrene, indeed, always appears under the same circumstances as typhus, viz., over-crowding and deficient ventilation; and it is possibly due to the same poison. From what has been stated, it is obvious, that surgical cases ought never to remain in the same ward with cases of typhus.

7. *Accidental Eruptions.* On several occasions, I have known numerous boils break out during convalescence, and prove very troublesome. In other cases, I have seen bullæ, filled with light or dark fluid, appear on various parts of the body, during the progress of the fever. Stokes has observed vesicles of this description, followed, after bursting, by deep ulcers, with sharp margins, as if punched out with an instrument.[a]

8. *Diffuse Cellular Inflammation,* ending in purulent infiltration, is an occasional complication or sequela. Its chief seat is the lower extremities. Its main symptoms are frequent rigors and perspirations, fever, great derangement of the stomach and bowels, prostration, sleeplessness and pain in the affected part. I have seen several examples of this complication, and others are recorded by G. A. Kennedy,[b] Graves,[c] etc.

9. *Inflammatory Swellings* or *Buboes* are not uncommon complications of typhus. Their most frequent sites are the parotid and submaxillary regions, and then they are usually attributed to inflammation of the glands; but, as was shown by Drs. Craigie[d] and Graves,[e] the inflammation has its seat mainly in the subcutaneous

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[u] *Chelius' Surgery*, South's ed., i. 56.
[y] *Mém. de Chir. Milit.* ii. 331.
[a] Stokes, 1854, xxix. 423.
[c] Graves, 1848, i. 261.
[e] Graves, 1848, i. 194.
[x] Jacquot, 1858, p. 211.
[z] Barrallier, 1861, p. 96.
[b] G. A. Kennedy, 1838, p. 35.
[d] Craigie, 1837, p. 301.

areolar tissues, and not in the substance of the glands. The pus, however, often insinuates itself between the lobules of the gland, which, after death, may be unusually dense, and have the appearance as if dissected out, while, on microscopic examination, the glandular tissue is found loaded with oil. Large portions of the subcutaneous areolar tissue may slough, and occasionally circumscribed drops of pus, with a small central slough, are found in the soft parts, at the circumference of the swelling.

The swellings in the parotid and submaxillary regions usually appear, towards the termination of the primary fever; but, in several instances, I have met with them in the first week, while in others, they are not developed until convalescence. They occur at almost every age, from 4 up to 70; but, as a rule, the patients have been above the average age of typhus cases (*i.e.*, upwards of 29. See page 62.) They are usually accompanied by considerable redness, tension, pain, tenderness, and sometimes œdema, of the superimposed skin; by inability to open the mouth, or to protrude the tongue; occasionally by dysphagia and deafness; and, in most cases, by great prostration and aggravation of the general symptoms. They often form with great rapidity: at one visit, the face may be natural; at the next, a few hours after, one side of it may be enormously swollen. They also advance rapidly to suppuration, an extensive collection of matter forming, in from two to four days; at other times, they recede without suppurating; or the swelling, after receding and almost disappearing, returns and rapidly advances to suppuration; occasionally, they co-exist with erysipelas of the face. When not opened artificially, they may burst externally, or into the mouth, or into the meatus of the ear. When they burst into the mouth, the breath, for some days after, is most offensive.

These inflammatory swellings may occur on one, or both sides of the face, and they are not restricted to this part of the body. I have met with them in the axilla, the groin, the mamma, the arms, thighs, and legs. As a rule, they do not exceed one, two, or three, in number; but occasionally, they are more numerous. I had lately under my care a case of typhus, complicated with numerous (about 20) subcutaneous abscesses, varying in size from a hazel-nut to a man's fist, in every part of the body. Some of them burst, and formed extensive gangrenous ulcers, and the patient sank from the profuse discharge. Dr. Stokes also records a case of typhus, in which 'large and foul buboes formed in various parts, and suppu-

'rated.'[f] On several occasions, I have known these swellings take the form of carbuncles.

Some writers have regarded these swellings, as critical and auspicious;[g] but, according to my observation, they are a most formidable complication, in every case where they advance to suppuration. It is true that they are occasionally met with in mild cases, about the period of crisis; but now and then, they are seen in the first week of the disease, and as a rule, they add greatly to the severity of the case, if they be not the immediate cause of death.

During the two years 1856-7, 21 cases of typhus in the London Fever Hospital were complicated with parotid swellings, of which 14, or 66·6 per cent., died; while the average mortality of all the remaining cases of typhus (1315) during the same period was only 20 per cent. This comparison is, perhaps, scarcely fair, as most of the patients with parotid swellings were above the average age of the other cases, and the mortality from typhus increases with age. Still, the average age of the 21 cases was 41 years, and during the ten years 1848-57, as well as the two years 1856-7, the rate of mortality of all the cases of typhus (including the parotid cases) between 40 and 50 years of age, was only 35 per cent. Again, during the first six months of 1862, 23 cases of typhus, complicated with parotid and other swellings, occurred at the London Fever Hospital, of which 9, or 40 per cent., were fatal, the mortality in the remaining 1084 cases being only 20 per cent. Here, the patients were not much above the average age, and several were children.

Parotid buboes and other inflammatory swellings have been noticed, in many epidemics of typhus, and have been usually regarded, as a serious complication. Many years ago, Riverius, in his account of an epidemic of typhus at Montpellier, stated that a number of the patients had swellings of the parotid region, appearing about the ninth or eleventh day, and that the majority of these cases proved fatal within two days.[h] According to Lind, many of the French prisoners at Winchester, in 1762, laboured under a very malignant form of typhus, 'attended with buboes 'both in the groin and arm-pits, and other pestilential symptoms.' He adds, that at Haslar Hospital, although he had never seen 'fevers rise to such a malignant height, as to produce buboes in 'the groin,' he had observed 'a swelling of the parotid glands,'

[f] Stokes, 1854, and Lyons, 1861, p. 193. [g] See Christison, 1840.
[h] Riverius, 1690.

and that 'such as were in this manner seized, commonly died.'[i] Swellings, in the groin and parotid region, were noted by Dr. Monro, in the typhus which prevailed in the British army in Germany, in 1761.[k] Parotid swellings were also observed in the *typhus siderans* of Saragossa, Torgau, and Mayence,[l] and in his account of typhus at Dantzic, M. Tort says, 'dans quelques cas aussi, mani-'festation de parotides ; toujours alors mort.'[m] Parotid swellings were a common complication of typhus, in the French army in the Crimea: 'ces parotidites,' says Jacquot, 'uniques ou doubles, sont 'toujours très graves.'[n] Lastly, M. Barrallier met with inflammatory swellings in the parotid and submaxillary regions, in 82 out of 1068 cases of typhus, and adds : 'La suppuration étendue 'des parotides, et du tissu cellulaire environnant, a souvent été d' 'un fâcheux augure ; sur les 24 malades, qui ont présenté cet 'accident (parotides suppurées), 15 ont succombé.'[o]

Inflammatory swellings in typhus are interesting, as they constitute a connecting link between this disease and Oriental plague. The more the subject is studied, the more the conviction is forced on the mind, that there is a strong analogy between the two diseases in question, in their causes, as well as their symptoms, and that, in fact, typhus is probably the plague of modern times.

In the first place, the two diseases resemble one another in their symptoms. The main differences are three, viz. : the more rapid progress of plague ; the presence in plague of buboes or inflammatory swellings in the inguinal, axillary, cervical, parotid, and submaxillary glands ; and the presence in typhus of an eruption, the spots of which have a tendency to become converted into petechiæ. But first, it has been shown that typhus may be as speedily fatal as true plague. (See pages 179, 211). Secondly, typhus is occasionally, like plague, complicated with buboes, which greatly aggravate the severity of the case. It is true, that these buboes appear later in typhus than in plague, and that they are met with in other febrile diseases, such as remittent and pythogenic fevers. But, although they are not pathognomonic either of plague or typhus, they are, as far as my knowledge extends, much more common in typhus, than in any other febrile diseases, excepting plague, while in the *typhus siderans* of Torgau and Mayence, they seem to have appeared as early as in plague. Thirdly, most writers agree in stating, that 'dusky-red, or pale, purplish spots, 'which, as the disease advances, acquire a livid hue,' are very

[i] Lind, 1763, p. 90.
[k] Monro, 1764.
[l] De Claubry, 1838, ed. 1844, pp. 33, 43, 45.
[m] Ib. p. 42.
[n] Jacquot, 1858, p. 211.
[o] Barrallier, 1861, p. 254.

common in plague.[p] Among the '*Directions for the Searchers*,' drawn up by the Royal College of Physicians of London, in 1665, is the following: 'Whether there be any tokens, which are spots 'arising upon the skin, chiefly about the breast and back, but 'sometimes also in other parts; their colour is something various, 'sometimes more reddish, sometimes inclining a little towards a 'faint blue, and sometimes brownish, mixed with blue.'[q] Many observers have been struck with the similarity, in the symptoms, of typhus and plague. The early writers often confounded the two diseases (*pestis* and *febris pestilens*), while both Cullen and Sauvages regarded plague, as merely a severe form of typhus.[r] Sydenham, speaking of typhus (*febris pestilens*), says: 'Cum ipsissima peste specie convenit, nec ab ea, nisi ob gradum 'remissiorem, discriminatur.'[s] The historians of the outbreak of plague, at Marseilles, in 1720, observe: 'La rapidité et quelques 'accidents sont les seules choses, qui, distinguent les fièvres 'malignes ordinaires de la peste.'[t] Dr. Ferriar wrote as follows: 'Although the symptoms of eruptions and buboes be distinguished 'by individual characters in the plague, yet they do not depart, in 'their general type, very far from the symptoms of malignant 'fevers; for the latter are very commonly attended by flat erup-'tions, which physicians call petechiæ, and glandular abscesses are 'not unfrequent in them.'[u] According to Dr. Copland, the symptoms of plague 'differ but little from those of true typhus 'fever, excepting in the appearance of carbuncles and buboes.'[x] Lastly, the celebrated Egyptian physician, Clot Bey, on visiting the London Fever Hospital, some years since, was much struck with certain cases of typhus, complicated with swellings in the parotid region, and declared that, in Egypt, they would be regarded as examples of plague. Excepting the buboes, the post-mortem appearances of typhus and true plague are identical.[y]

But secondly, in the plague, as in typhus, there is reason to believe, that the poison can be generated spontaneously, and that the disease does not of necessity arise from contagion or from some epidemic influence. On this subject, the reader is referred to the works of Heberden,[z] and Hancock,[a] and to the valuable report on the Plague and Quarantine, drawn up by a Commission of the French Royal Academy, in 1846, and published in the name of Dr. Prus.[b] From the evidence, collected in these works and else-

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[p] See article 'Plague,' in *Lib. of Med.* vol. i. 1840, p. 192. [q] HEBERDEN, 1801.
[r] *Typhus Ægyptiacus* (SAUVAGES); a variety of *Typhus gravior* (CULLEN).
[s] *Op. Om.* Syd. Soc. Ed. p. 96. [t] HANCOCK, 1821. [u] FERRIAR, 1810, i. 268.
[x] COPLAND, 1858, iii. 196. [y] CRAIGIE, 1834, p. 273. [z] HEBERDEN, 1801.
[a] HANCOCK, 1821. [b] PRUS, 1846.

where, it seems probable, that the poison of plague is generated by the concentration of animal exhalations, consequent on overcrowding with deficient ventilation. In Cairo, the modern head quarters of the plague, the streets are extremely narrow, and the population is crowded into close chambers, quite devoid of all ventilation. Throughout the rest of Egypt, the habitations are no better; the house, or rather the hole, of the Egyptian, is built of mud, and the door is so small and low, that it can only be entered by creeping. A number of these huts, which resemble so many ant-hills, are constructed close to one another, and every means of ventilation is cut off, while whole families lie huddled together. Such are the localities in which plague appears, independently of any importation from without. Moreover, the great predisposing cause of plague, as of typhus, is starvation. Failures of the crops, and other causes of famine, convert sporadic cases of plague into great epidemics. Speaking of the events which preceded the great epidemic of plague in the fourteenth century, Hecker observes: 'Children died of hunger 'in their mother's arms. Want, misery, and despair were general 'throughout Christendom."[c] "The outbreak of the plague" says Dr. Milroy, in his review of the French Report, 'has not un-'frequently followed upon wars, famines, and other wasting 'calamities; and, on the other hand, its ravages have invariably 'been observed to become less frequent and less desolating, in 'proportion as the condition of the inhabitants of the affected 'countries, in point of civilisation and comfort, has improved.[d] According to M. Prus, 'Si nous recherchons, avec soin, les causes 'qui paraissent exercer l'influence la plus grande, sur le développe-'ment de la peste, nous pourrons les résumer ainsi: habitation sur 'des terrains d'alluvion ou sur des terrains marécageux; *maisons 'basses mal aérées, encombrées; air chaud et humide;* action des 'matières animales et végétales en putréfaction; *alimentation 'malsaine et insuffisante; grande misère physique et morale."*[e] The resemblance between the causes of plague and typhus requires no comment. It is probable, that the warm, moist climate of Egypt may lead to the development of plague from an amount of overcrowding, etc., which, in this country, would only suffice to generate typhus. But some centuries ago, when our dwellings resembled those of the Egyptians, plague was a common disease in London, and occasionally, like typhus, it appeared in great epidemics. It has been the fashion, to refer the origin of these

[c] Hecker, 1844, p. 17. [d] Prus, 1846. [e] Ib.

epidemics to imported contagion; but there is no satisfactory evidence, that this was the case. If the poison of plague was always imported, it is strange that during the last two centuries, while an extended commerce has increased the means of importation a thousand-fold, plague (except in the form of typhus) has been unknown in Britain. No one will be bold enough, to attribute this exemption to the operation of our Quarantine laws.

The disappearance of the plague from London was coincident with an improved construction of the dwelling-houses, which followed the great fire of 1666. Heberden describes the state of the city, prior to the fire, as follows: 'The streets were narrow and 'crooked, and many of them unpaved; the houses were built of 'wood, and lofty; they were dark, irregular, and ill-contrived, 'with each story hanging over the one below, so as almost to meet 'at the top, and thereby preclude, as much as possible, all access to a 'purer air; they were besides furnished with enormous signs, 'which, by hanging into the middle of the street, contributed not 'a little, to prevent all ventilation below.'[f] 'It is probable,' says Hancock, 'that if this country has been so long forsaken by the 'plague, as almost to have forgotten, or at least to be unwilling 'to own, its natural offspring, it has been because the parent has 'been disgusted with the circumstances, under which that hateful 'birth was brought to light, has removed the filth from her doors 'in which it was matured, and has adopted a system of cleanliness 'fatal to its nourishment at home. But if ever this favoured 'country, now grown wise by experience, should relapse into 'former errors, and recur to her odious habits, as in past ages, it 'is not to be doubted that a mutual recognition will take place, 'and she will again be visited by her abandoned child, who has 'been wandering a fugitive among kindred associates, some-'times in the mud cots of Egypt, sometimes in the crowded 'tents of Barbary, and sometimes in the filthy kaisarias of 'Aleppo.'[g]

Moreover, many epidemics of plague in Europe, have been preceded and accompanied by a great prevalence of typhus. Instances of this nature have already been referred to (pages 23, 24, 25), and others will be found in the works of Heberden and Hancock. Many writers state, that the one affection merged into the other, so that it was sometimes difficult to say, whether a case was typhus, or genuine plague.

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[f] HEBERDEN, 1801. [g] HANCOCK, 1821.

Case XX.

Typhus complicated with Parotid Swellings—Recovery.

John F——, aged 12, admitted August 4th, 1856. Ill a week, and delirious, for two nights, before admission.

August 5th (8th day.) Pulse 117, small and soft; skin hot and dry; well-marked typhus eruption. Tongue dry and brown, along centre. Sordes on teeth; bowels confined. Has a heavy, confused expression; face dusky; slept at intervals during the night; much delirium. Ordered, carbonate of ammonia, wine, (6 ounces), milk, and beef-tea.

August 7th (19th day). Pulse 100. Bowels opened twice from castor-oil. Other symptoms as before, except that there is slight swelling and tenderness, without any hardness, over both parotids. Poultices were ordered.

August 8th (11th day). Swellings increased, especially that on right side, which is slightly red on surface, and somewhat hard and tender. Swallows well.

August 9th (12th day). Pulse 100, and regular; skin dry and cool; rash still out; swellings larger, hard, and painful; much redness of skin below left ear; tongue moist and furred, protruded with difficulty; but swallows well. One stool. Slept better, and less delirium.

August 13th (16th day). Pulse 120, and weak; rash almost gone; purulent discharge from both ears, but swellings still hard, painful, and not pointing; tongue moist, and some improvement in general symptoms, but is very prostrate. Ordered quinine, milk, arrowroot, beef-tea, and one egg.

August 15th (18th day). Both ears have been discharging freely, and to-day, both swellings are soft and pointing. They were both opened by a free incision. A quantity of pus escaped, and on the 21st, a large slough came away from the opening on the right side. After the evacuation of the matter, the patient convalesced rapidly, and on the 23rd, the openings had ceased to discharge.

Case XXI.

Typhus complicated with Bronchitis, Pneumonia, and Inflammatory Swelling in Left Parotid region. Death on the 27th day.

Frederick G——, aged 34, admitted into the London Fever Hospital, April 7th, 1862, having been taken ill, on the 3rd, with rigors, headache, and general pains. On admission, the pulse was 108; the headache was intense; the patient was confused, very restless, and slept badly; the skin was covered with a typhus-rash. Cold lotions, mineral acids, and beef-tea were prescribed.

On the 9th day, the headache had ceased, but the patient was very delirious, getting out of bed; the rash was very abundant and darker. Wine (4 ounces), and the morphia and antimony draught were ordered, after which he slept.

On the 11th day, more prostrate and almost unconscious. Tongue dry and brown; pulse 116, feeble; urine and fæces in bed. Brandy was substituted for wine.

On the 14th day, pulse 102; very prostrate; much quiet delirium; takes notice when spoken to, and that is all; eruption copious and petechial. The brandy was increased to 10 ounces.

On April 19th (16th day), the left parotid region became enormously swollen in a few hours, and very painful. The prostration increased; the pulse was 120, and with difficulty felt; the tongue and hands were tremulous; the swelling was painted with a strong solution of nitrate of silver, and covered with cotton wool. Liquor cinchonæ, sulphuric ether, and ten ounces of brandy were prescribed, with the addition, after two days, of three eggs, and two pints of porter.

April 25th (22nd day). The swelling has increased considerably, and to-day a small opening has formed behind the ear, from which a very little pus has escaped. The skin over the entire swelling pits on pressure, but there is no distinct fluctuation. Has no cough, but the breathing is hurried, the lips slightly livid, and sibilant râles are audible, all over front of chest; the rash has quite disappeared; no albumen in urine. Sinapisms to the chest, and a mixture containing ammonia, ether, and senega, were prescribed.

On April 28th, there was considerable discharge from the opening, and the swelling was smaller and less tense; pulse 130, and feeble; respirations, 68; face, livid; no cough or expectoration, but moist râles audible over whole of right lung, and on back and side of left lung; both lungs dull on percussion, posteriorly; face livid; swallows well, but is scarcely conscious. Sinapisms to the chest, 12 ounces of brandy, and a mixture containing 15 minims of sulphuric ether and turpentine, every two hours, were prescribed.

No improvement took place, and death occurred on April 30th.

Autopsy, 24 hours after death.

On laying open the swelling, the whole of the subcutaneous areolar tissue was found to be in a state of slough; the lobules of the parotid were unusually hard, and, as it were, dissected out and bathed in the puriform fluid. In the muscles and other tissues, near the circumference of the swelling, were a number of circumscribed collections of pus, not larger than a pea, with a small central slough in each. The glandular tissue of the parotid contained an immense amount of oily matter.

Bronchial tubes filled with frothy mucus; great hypostatic consolidation of both lungs; granular consolidation of lower sixth of right lung. Both sides of heart filled with dark coagulum and fluid blood.

Intestines healthy; liver somewhat friable; spleen $6\frac{1}{2}$ ounces, rather soft. Right kidney, $4\frac{3}{4}$ ounces; left, $5\frac{1}{4}$ ounces; structure, normal.

i. Other Specific Diseases.

Hunter's doctrine,[h] that no two of the so-called specific diseases can co-exist in the body, has been disproved by modern observation. Abundant evidence has been collected, to show that any two of these diseases may run their course together, both eruptions, in the case of the exanthemata, being present at one time. A *résumé* of this evidence will be found in the British and Foreign Med. Chir. Review for July, 1859.[i] The co-existence of typhus with other specific diseases, however, still requires investigation. The following observations bear on the question:—

1. *Typhus and Variola.*—Barrallier, on the authority of several French naval surgeons, mentions a number of cases, where typhus and variola ran their course together in the same persons.[k] A similar case was lately observed at the London Fever Hospital, under the care of my colleague, Dr. Buchanan.

Case XXII.

Case illustrating Co-existence of Variola and Typhus.

A girl, aged 15, was seized on June 1st, 1862, with severe pains in the back, vomiting, and loss of appetite, followed by an eruption of variolous papules on June 3rd. On June 6th, she was removed to the Small-pox Hospital, where the symptoms ran the usual course of a mild attack of variola, modified by vaccination. There were good cow-pock marks on the arm. The febrile symptoms, however, did not recede, and, on June 11th, a typhus-rash made its appearance on the trunk. On June 12th, she was removed to the Fever Hospital; and at this date, there were a number of desiccating pustules on the face, together with a well marked typhus-rash on the chest and abdomen. This rash was still distinct on June 18th, but disappeared on the following day, and the patient made a good recovery. Several small-pox cases had occurred, in the next house to that where the girl was taken ill, and there was also much typhus in the neighbourhood. The girl had also been removed to the Small-pox Hospital in a carriage used to convey typhus patients.

2. *Typhus and Scarlatina.*—Although I have never seen the eruptions of these two diseases actually co-existing, I have repeatedly known the one follow close on the other. I have notes of four cases, where scarlet fever appeared within a fortnight of the commencement of convalescence from typhus; and in one, the scarlet rash came out on the seventh day after the disappearance of the typhus rash. I have also notes of seven cases of typhus succeeding scarlet fever, in two of which the attack of typhus commenced on

[h] Hunter's *Works*, Palmer's ed. i. 313; iii. 4.
[i] Murchison, 1859 (No. 4). [k] Barrallier, 1861, p. 42.

the third or fourth day of convalescence from scarlet fever, while the cuticle was desquamating, or before the poison of scarlet fever was entirely eliminated from the system. In one of the two cases, extensive anasarca, lumbar pain, and scanty, albuminous, smoky urine were observed towards the termination of the attack of typhus.

3. *Typhus and Pythogenic Fever.*—Evidence, as to the occasional co-existence of these two fevers, will be found in a subsequent part of the volume.

Sect. IX. Varieties of Typhus.

AUTHORS have described different varieties of typhus, depending on the severity of the disease, the prominence of certain symptoms, the presence of complications, and the circumstances in which the fever originates. The comparative frequency of some of these forms varies in different epidemics; but this is probably due to differences in the constitution and habits of the patients, and to the circumstances under which the epidemic arises, rather than to any change in the constitution or type of the fever itself. The following varieties result from the prominence of certain symptoms.

1. *Inflammatory Typhus.* This designation has been applied to those cases, where there is great febrile reaction, much heat and flushing of skin, severe headache, and often acute delirium. This form is chiefly observed in the young and robust, and in persons of the upper class. It occurred in only 40 out of 1,302 cases observed by Barrallier. Most of the cases of Inflammatory Continued Fever, or Synocha, described by different writers, have probably been examples of Relapsing Fever, or of acute inflammations.

2. *Nervous or Ataxic Typhus* is that form, in which nervous symptoms, such as delirium, somnolence, tremors and subsultus, predominate. The eruption is usually dark and petechial. Such cases have also been designated *Typhus Comatosus* and *Brain-Fever.* This form occurred in 109 out of 1,302 cases observed by Barrallier.

3. *Adynamic Typhus* is characterized by the early supervention of marked asthenic symptoms — great prostration, involuntary evacuations, impairment of the heart's action, and tendency to collapse. The skin may be cool and the pulse slow. I have known patients pass through the entire attack, in a state of prostration approaching to collapse, with the mind little, if at all, affected. Barrallier noted the adynamic form in 92 out of 1,302 cases. Most commonly, the adynamic and ataxic forms are combined, constituting—

4. *Ataxo-adynamic Typhus,* or the *Congestive Typhus* of Armstrong.

This is by far the most common form of Typhus. It was observed by Barrallier, in 810 out of 1,302 cases.

5. *Typhus Siderans.* This term has been applied to those cases, already alluded to (page 178), where the disease has proved fatal within a few days, or sometimes hours, of its commencement.[l]

6. *Mild Typhus.* Cases are met with, particularly at such times and places, as the disease is not epidemic, in which the fever is of short duration and runs a mild course, without severe symptoms of any sort. The fever was of this mild character, in 235 out of 1,302 cases, observed by Barrallier. Were it not for the eruption, these cases would often be regarded as examples of simple fever or febricula. Mention is made of this form by Hildenbrand, under the appellation of *Typhus levissimus.*[m]

Jacquot has described, under the head of *Typhisation à petite dose,* certain symptoms, such as malaise, slight fever, loss of appetite, gastric derangement, fatigue, headache, disturbed sleep, and occasional confusion of the mental faculties, which are observed in persons constantly exposed to contagion, without passing into actual typhus. True typhus sometimes supervenes upon this condition; but in some instances, this state lasts for several weeks, and ceases on removal from the typhus-atmosphere.[n]

Among the varieties dependent on the presence of peculiar complications, may be mentioned:—

7. *Catarrhal Typhus.* This is a common designation of typhus in Ireland, owing to its frequent complication with bronchitis.

8. *Scorbutic Typhus.* (See page 190).

9. *Glandular Typhus.* (See page 200).

10. *Dysenteric Typhus.* (See page 195).

Lastly, there are certain varieties of typhus, according to the localities where it prevails:—

11. *Jail Fever.* (See page 100).

12. *Ship Fever.* (See page 106).

13. *Military or Camp Fever.* (See page 108),

14. *Hospital Fever.* (See page 110).

Sect. X. Diagnosis of Typhus.

BEFORE the appearance of the eruption, the diagnosis of typhus must always be doubtful. The most characteristic symptoms are, pains and aching in the limbs, headache, a feeling of prostration and lassitude, chilliness, loss of appetite and furred tongue.

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[l] For an account of *Typhus siderans* see De Claubry, 1838 (ed. 1844), pp. 35, 43, 45, 119; Jacquot, 1858, p. 19.

[m] Hildenbrand, 1811, p. 113.

[n] Jacquot, 1858, p. 212.

If a person who has been exposed to the poison of typhus is attacked by these symptoms, the diagnosis is tolerably certain. All doubt is removed on the appearance of the eruption.

Many diseases may, in their advanced stages, assume a typhoid character, and differ only from typhus in the absence of the peculiar eruption. The diseases, however, with which typhus is most readily confounded, are, relapsing fever, the so-called typhoid or pythogenic fever, some forms of remittent fever, purpura, measles, meningitis, delirium tremens, pneumonia, disease of the kidneys, pyæmia, and other blood-poisonings.

1 and 2. The distinctions between typhus and the *Relapsing and Pythogenic Fevers* will be best considered, after the symptoms of these fevers have been described.

3. *Remittent Fever.* The remittent fevers of this climate can never be mistaken for typhus; but certain forms of tropical remittent fever, known as 'typhoid or malignant remittents' and 'jungle fever,' occasionally present symptoms having a close resemblance to those of typhus, such as a small soft pulse; dry, brown, retracted tongue; dorsal decubitus and great prostration; low muttering, delirium; tremors and subsultus; contracted pupils, and even petechiæ. Some years ago, I had an opportunity of seeing many such cases in Burmah; and it is a fact, that they have often been described as examples of true typhus in the tropics. In distinguishing the two diseases, the circumstances in which each is wont to appear, should be borne in mind. Typhus results from contagion or overcrowding; remittent fever results from malaria and is non-contagious. Typhus is rare in those countries where remittent fevers, of the character described, prevail; and in countries where the two diseases have prevailed together, as in the Crimea, typhus is most common in the winter and spring, remittent fever towards the end of summer and in autumn. True remissions are not met with in typhus; and the exacerbations which occur are usually nocturnal, whereas those of remittent fever are for the most part diurnal. The great solid enlargement of the spleen, so often noticed in malarious fevers, is not characteristic of typhus, while the peculiar eruption of typhus is never met with in remittent fever. Lastly, quinine, which is often a specific in malarious fevers, has no beneficial effect in typhus.

4. *Purpura.* Although Riverius long since distinguished purpura (*'petechiæ sine febre'*) from the petechiæ of typhus (*'febris petechialis'*), the two affections have sometimes been confounded. The non-contagious character of purpura; the absence of pyrexia; the characters of the spots, which are larger than the petechiæ of

typhus, and are not preceded by the characteristic typhus-rash; the occurrence of hæmorrhage from the gums, nose, bowels, and other mucous surfaces; the blanched countenance, and the absence of cerebral symptoms, are characters which usually suffice to distinguish purpura from typhus. At the same time, it must be borne in mind, that when typhus is complicated with scurvy, purpura-spots, vibices, and hæmorrhages from the mucous surfaces may be superadded to its ordinary symptoms. This is, perhaps, the real nature of the *purpura febrilis* described by Dr. Copland, and other writers.[o]

5. *Measles.* Typhus, in children, may at first be readily mistaken for measles, from the similarity of the two eruptions, which in both cases appear about the fourth day. The eruption of measles, however, is of a brighter tint, and does not pass through the different stages observed in that of typhus; it differs also from that of typhus, in being preceded by sneezing and other catarrhal symptoms. The diagnosis may be assisted, by examining other members of the same family who may be affected at the same time. Measles is almost invariably confined to the children of a family; whereas typhus rarely attacks children, before the adult members of the family.

6. *Meningitis; Encephalitis.* At the commencement of this century, the symptoms of typhus were referred to cerebral inflammation; and, at the present day, typhus is not uncommonly designated 'Brain-Fever.' The chief points of distinction between typhus and inflammation of the brain and membranes, are the following. In inflammation, the headache is much more intense, and of a throbbing, darting, bursting, or constricting character; in typhus, the patient rarely describes it by such terms. The delirium of inflammation is more violent and acute than that of typhus, and accompanies, or alternates with, the headache; whereas the headache has almost always ceased in typhus, before the delirium commences; the loud cries and screams observed in the delirium of meningitis do not occur in typhus. In inflammation, there is great intolerance of light and sound; but in typhus the senses are obtuse, and deafness is more common. In both diseases the face is flushed and the conjunctivæ are injected; but in typhus the flush is more dusky, and the blood in the conjunctival vessels of a darker tint than in inflammation. In both diseases, there may be general convulsions followed by coma, but typhus never commences in this way, as meningitis sometimes does. Strabismus

[o] *Med. Dict.* iii. 553.

and partial palsy are far more common in inflammation than in typhus. The physiognomy of meningitis is anxious and expressive of pain, or wild and defiant; in typhus, it is oftener blank and stupid. In typhus, there is much more muscular prostration from the first than in inflammation. The pulse in inflammation is usually firm; in typhus, it is soft and compressible. Nausea and urgent vomiting are common in inflammation; rare, in typhus. Lastly in typhus, there is the peculiar eruption appearing about the fourth or fifth day.

But the diagnosis is not always so easy as might be imagined. The *delirium ferox* of typhus (see page 153), often closely simulates inflammation; and in such a case, the presence of the eruption, or the exposure of the patient to the poison of typhus, can alone assist us in distinguishing this disease from meningitis. When the rash of typhus is present, it may always be concluded that there is no cerebral inflammation, for *post-mortem* examinations show that inflammation of the brain or of its membranes rarely, if ever, occurs, even as a complication of typhus. Stokes has well observed, that those symptoms which indicate inflammation of the brain under ordinary circumstances, do not indicate inflammation, when the case is one of typhus fever. When the rash is absent, the diagnosis must sometimes be doubtful. The stage of collapse in cerebral inflammation is also indistinguishable from the typhoid state of typhus, in the absence of eruption or of any history of the case.

7. *Delirium tremens.* The delirium of typhus may often be justly designated delirium tremens (see page 152). How then are we to distinguish the delirium tremens of the drunkard, from that of typhus? In the former, the tongue is moist and covered with a creamy fur, and not dry and brown, as in the delirious stage of typhus; the skin is moist, and there is no eruption; the mode of accession is also different; there are no rigors, headache, or general pains, but the affection commences with loss of sleep and delirium. Lastly, the circumstances preceding, and giving rise to, an attack of delirium tremens, will seldom leave any doubt as to the nature of the case.

8. *Pneumonia.* Latent pneumonia is not unfrequently confounded with typhus. In asthenic or typhoid pneumonia (where the apex is often the part of the lung first and chiefly implicated), the symptoms of the local disease may be entirely masked by those of a general typhoid condition. I have known many cases of this nature sent to the Fever Hospital as examples of typhus, whether the latter disease was prevalent or not. When a patient is seen for

the first time in a typhoid condition, and when no eruption can be detected on the skin, the medical attendant should never fail to make a careful examination of the lungs. If signs of pneumonia be discovered, and especially if they be situated at the apices of the lungs, the typhoid symptoms may be ascribed to the local lesion, unless the patient has been exposed to the poison of typhus, and then the diagnosis must be doubtful, and it is well to act on the supposition, that the patient is labouring under an infectious disease.

9. *Diseases of the Kidney.* From what has already been stated, it is not surprising that uræmia from renal disease is apt to be mistaken for typhus; indeed, it is sometimes impossible to distinguish the two diseases. The dry brown tongue, stupor, contracted pupil, low muttering delirium, and all the characteristics of the typhoid state, belong to both. It has often happened, that cases of uræmia from kidney disease have been sent to the Fever Hospital as cases of typhus, where the absence of eruption has alone raised any doubt on the point. Moreover, in typhus, the urine may contain albumen and tube-casts, urea may be detected in the serum of the blood, and death may take place by convulsions and coma, although there has been no previous disease of the kidneys. The diagnosis is still further embarrassed by the circumstance, that, in those cases of renal disease (the contracted granular kidney), which most resemble typhus, there may have been no dropsy at the time of death, or at any period of the patient's life. This form of kidney disease, however, is often associated with gout, and hence in all doubtful cases, enquiries should be made as to whether there is any gouty history.

The following case shows how closely renal disease may simulate typhus:—

Case XXIII.

Uræmia, from Renal Disease, Simulating Typhus.

A man, aged 60, was admitted into King's College Hospital, under my care, in August 1858, with all the symptoms of the typhoid state,—a dry, brown, retracted tongue, great muscular prostration, drowsiness, low muttering delirium, contracted pupils. The pulse was 96, and feeble; there was no eruption on the skin, and no indication of pulmonary disease, and there was not a trace of œdema. All the history that could be obtained was, that the patient had been ill for only a week, and that his symptoms, before admission, had been anorexia and constipation, slight headache, loss of memory and mental confusion; he had suffered from several attacks of gout, but never had dropsy at any period of his life. The man died, at the end of a fortnight from the commencement of his illness.

For the last three days of his life, he was in a state of profound coma, but he had no convulsions. Unfortunately, no urine could be obtained for examination, as the small quantity secreted was passed involuntarily.

On *post-mortem* examination, the kidneys were found to be remarkably small, the two together weighing less than five ounces; their surfaces were granular, and the capsules adherent. The cortical substance was much atrophied and firm, and contained several cysts. Many of the uriniferous tubes were blocked up by deposits of urate of soda.

Many other cases of renal disease, simulating typhus, have come under my notice at the Fever Hospital, (see page 12); and others have been recorded by Dr. George Johnson.[p]

10. There are other *Blood-poisonings*, such as erysipelas, pyæmia, jaundice, glanders, etc., where symptoms may appear, like those of typhus; but these diseases have distinct characters, which can rarely leave any doubt, as to the nature of the case. At the same time, erysipelas, pyæmia, and jaundice may exist as complications of typhus. Speaking generally, it may be said, that the only certain means of distinguishing typhus from several other blood-poisonings, is the presence of the characteristic eruption. When this is present, typhus is to be regarded as the primary disease, and the erysipelas, pyæmia, etc., as secondary complications. But in simple typhus, the eruption may be absent, and there is no reason why it should not be also absent in complicated cases. Hence, in certain cases of uræmia, pyæmia, erysipelas, and typhoid jaundice, especially during an epidemic of typhus, it may be difficult to decide whether they are the primary diseases, or are to be regarded as complications of unspotted typhus.

Sect. XI. Prognosis and Mortality.

In forming a prognosis in typhus, we must take into consideration the rate of mortality, the circumstances which influence that rate of mortality, the presence and severity of certain symptoms and complications in individual cases, and the mode of fatal termination.

a. Rate of Mortality.

There are few data for calculating correctly the rate of mortality of typhus, as in most hospital reports, all forms of continued fever are classified along with it. It is obvious, that if cases of relapsing fever, which are seldom fatal, and cases of febricula, which always recover, be included with typhus, the mortality of the latter disease must be greatly reduced. The following results are free from such

[p] *Med. Times and Gaz.* Jan. 16th, 1858 p.53.

objections. Table X. shows the mortality among the cases of typhus admitted into the London Fever Hospital during 14½ years.

TABLE X.[q]

Years.	Admissions.	Deaths.	Mortality per cent.
1848	526	106	20·15
1849	155	39	25·16
1850	130	24	18·46
1851	68	6	8·82
1852	204	24	11·76
1853	408	90	22·06
1854	337	68	20·18
1855	342	82	24·
1856	1062	207	19·49
1857	274	69	25·18
1858	15	9	60·
1859	48	18	37·5
1860	25	10	40·
1861	86	16	18·6
1862 to June 30th	1107	232	20·95
Total	4787	1000	20·89
Deducting 79, who died within 24 hours . .	4708	921	19·56
Deducting 172, who died within 48 hours . .	4615	828	17·94

Thus, out of 4787 cases of typhus, 1000 died, making a mortality of 20·89 per cent., or of 1 in 4·78. But a large number of the patients were moribund, on admission. Deducting those fatal within twenty-four hours after admission, the mortality falls to 19·56 per cent.; and deducting those fatal within forty-eight hours, it is only 17·9 per cent., or about 1 in 5⅗. In one-half of the years, the mortality exceeded the average; and in one year, it was under 9 per cent. Although a large proportion of the patients in the London Fever Hospital are the aged and infirm inmates of the metropolitan workhouses, this mortality from typhus does not exceed what has been observed elsewhere. Out of 108 cases admitted into King's College Hospital under the late Dr. Todd, between the years 1840 and 1858, 27 died, or the mortality was 25 per cent.[r] In the Edinburgh Infirmary, of 538 cases admitted under the care of Dr. W. Robertson,[s] in the year 1847-8, 133, or 24·72 per cent. died, and of 539 cases under Dr. Paterson, 111 or 20·6 per cent. died[t]; of 363 cases admitted into the same institu-

[q] In this and the other tables in this work, the deaths for each year have reference only to the patients *admitted* in that year. A patient admitted in December, 1851, and dying in January, 1852, has been entered as a death in 1851.

[r] *Brit. and For. Med. Chir. Rev.* Oct. 1860, p. 332. [s] ROBERTSON, 1848, p. 370.

[t] R. PATERSON, 1848.

tion in 1848-9, 80, or 22·3 per cent. died.[u] Of 9,485 cases admitted into the Glasgow Infirmary during eleven years (1843-53), 1,700, or 18 per cent. died[v]; and of 1,402 cases, during five years (1857-1861), 236 died, or 16·83 per cent. Of 1,370 cases admitted into the Barony Parish Fever Hospital at Glasgow, in 1847-8, 236, or 17·23 per cent., died.[x] Adding together all the above results, we have 18,592 cases, and 3,523 deaths, or the mortality is 18·78 per cent., or 1 in 5·27.

It may, therefore, be assumed that one out of every five persons attacked by typhus will die. This appears to be the average rate of mortality in England and Scotland; and although the mortality from fever has always been noted as much smaller in Ireland, I know of no data for concluding that the mortality from maculated typhus is less, than that above stated. Of 1,366 cases under Dr. Reid of Belfast, in 1847, there died 258, or 19 per cent.[y] According to Dr. Lyons, the mortality in most of the Irish epidemics, has been one in three, or sometimes higher.[z] It is obvious, therefore, that typhus is always a very dangerous disease.

b. Circumstances influencing the Rate of Mortality.

1. *Times and Seasons.*—The mortality from typhus does not appear to be affected by the season of the year, as is shown by the following table, which gives the number of admissions into the London Fever Hospital during ten years (1848-1857):—

TABLE XI.

Seasons.	Admissions.	Deaths.	Mortality per cent.
Spring . . .	1069	218	20·39
Summer . . .	988	206	20·85
Autumn . . .	659	131	19·88
Winter . . .	790	160	20·25
Total	3506	715	20·39

But, as regards different years, the rate of mortality at the Fever Hospital varied greatly, without any reference to months or seasons. The mortality from typhus has sometimes been observed to be smallest, at those times when the disease has been least prevalent. Thus, in the year 1851, when only 68 cases were admitted into the London Fever Hospital, the mortality was only 8·82 per cent. Again, at Edinburgh, the mortality during the great

[u] *Statistical Tables*, 9th ser. p. 14.
[v] M'GHIE, 1855, p. 161.
[x] J. PATERSON, 1848, p. 357.
[y] *Irish Report*, 1848, viii. p. 297.
[z] LYONS, 1861, p. 215.

epidemic of 1847 was 1 in 4; but during the last few years, in which typhus has rarely been met with, the mortality, according to Dr. W. T. Gairdner, has not exceeded 3 in 45, or 1 in 15.[a] This observation, however, does not always hold good, and certainly has not applied to London of late years. Thus, in 1856, of 1062 cases of typhus admitted into the Fever Hospital, the mortality was under 20 per cent.; whereas, during the three years 1858-60, when the cases were extremely few, the mortality was 42 per cent. (See Table X.)

It has often been found, that the mortality has been greatest at the commencement and height of great epidemics, and that it has declined as the number of cases has diminished. This is well shown in the annexed table, which gives the admissions and mortality of typhus cases during five successive quarters, commencing in October, 1855:—

TABLE XII.

Date.	Admissions.	Deaths.	Mortality per cent.
October to December, 1855 .	143	35	24·5
January to March, 1856 . .	421	97	23·04
April to June „ . .	317	71	22·4
July to September „ . .	146	23	15·48
October to Dec. „ . .	178	16	8·98

A similar remark was made by Dr. Peacock,[b] with regard to typhus, in Edinburgh, in 1839, '40, and '41, and the same thing occurred at Edinburgh, in the great epidemic of 1847-8. This increased mortality may be accounted for in various ways;—by the circumstance, that the disease first attacks the aged and infirm, and the sufferers from want of food, who are least able to resist it; or by the rapid development of the epidemic, taxing the resources, and deranging the economy of hospitals, and so leading to overcrowding and deficient nursing. Still, it must be remembered, that the mortality is sometimes equally great, when the disease is far from being very prevalent.

2. *Sex.*—Most observations show that typhus is somewhat more fatal in males, than in females. Table XIII. gives the results at the London Fever Hospital, for ten years:—

TABLE XIII.

	Admissions.	Deaths.	Mortality per cent.
Males	1737	368	21·18
Females	1769	347	19·61
Males and Females . .	3506	715	20·39

[a] W. T. GAIRDNER, 1862, No. 2, p. 159. [b] PEACOCK, 1843.

In the Edinburgh Infirmary, during the years 1841-2, Dr. Peacock ascertained, that of 377 male cases of typhus, 69, or 18·3 per cent., died; whereas, of 371 females, only 45, or 12·12 per cent., died.[c] Again, in 1847, of 330 males affected with typhus at Edinburgh, under the care of Dr. W. Robertson, 87, or 26·36 per cent., died; whereas, of 208 females, only 46, or 22·11 per cent., died:[d] of 258 males under Dr. Paterson, 65, or 25·1 per cent., died; whereas, of 281 females, only 46, or 16·37 per cent., died.[e] Of 1,011 male cases at Glasgow in 1847, 328, or 32·44 per cent., died; but of 878 females, only 182, or 20·7 per cent. died.[f]

Taking all the cases of continued fever, admitted into the Edinburgh Infirmary during eleven years (1839-1850), of 10,811 males, there died 1,477, or 13·66 per cent.; while of 8,863 females, only 897, or 10·12 per cent., perished.[g] The mortality from fever in the Dundee Infirmary, in 1836 and 1837, was for females, 1 in 18, and for males, 1 in 11[h]; that in Glasgow in 1835-6, according to Dr. Cowan, was 1 in 6 for males, and only 1 in 11 for females.[i] Barker and Cheyne long ago expressed the opinion, that epidemic fever in Ireland was more fatal to men than to women,[k] and this statement has been confirmed by most subsequent observations.[l] In Stockholm, of 2,181 males affected with fever, 252, or 20·7 per cent., died; but of 1,005 females, the mortality was only 87, or 8·65 per cent.[m]

There can be no doubt then, that, as a rule, the mortality is greater among males. It is difficult to account for this. One explanation offered is, that the average age of the female patients is less than that of the males. Of Dr. Peacock's cases, the average age of the females was certainly slightly less than that of the males. But in the London Fever Hospital, the proportion of cases above thirty was much greater among females; of 1,742 females, 834, or 48·4 per cent. were above thirty, whereas of 1,714 males only 675, or 39·3 per cent., exceeded that age. The same remark applies to Dr. Cowan's cases. A more probable explanation is, that men are more likely to expend their muscular force during the early stages of the fever, by attempting to follow their ordinary avocations, and that a larger proportion of them have led intemperate lives.

3. *Age* exercises such a remarkable influence over the rate of mortality from typhus, that no just comparison between the rates of mortality, at different times and places, can be made

[c] Peacock, 1843. [d] Robertson, 1848, p. 370. [e] R. Paterson, 1848, p. 398.
[f] Steele, 1848, p. 161. [g] *Reports*, 10th ser. p. 20. [h] Bartlett, 1856, p. 256.
[i] Cowan, 1838. [k] Barker and Cheyne, 1821, i. 90. [l] Bartlett, 1856, p. 255.
[m] Huss, 1855, p. 58.

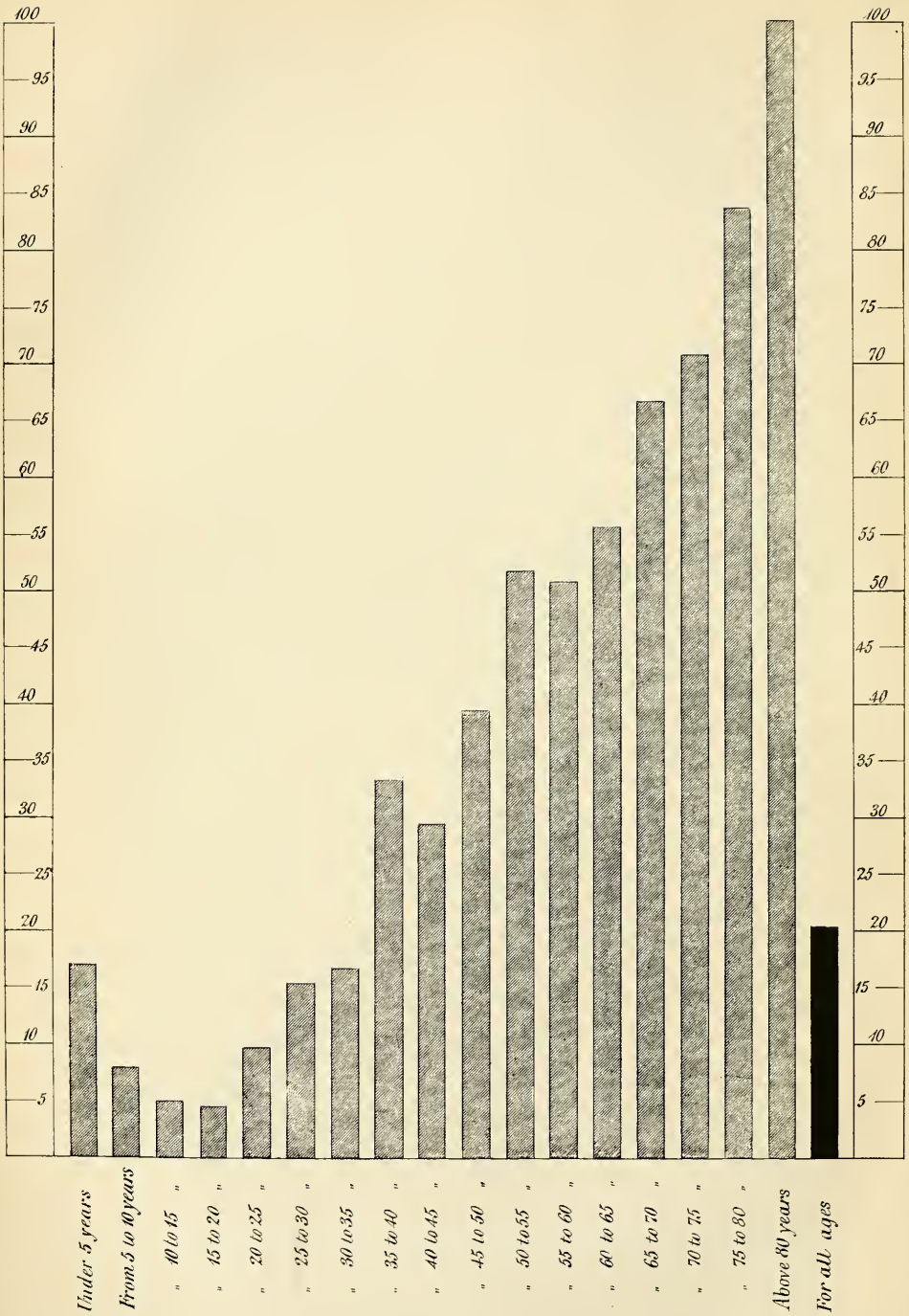

Diagram V, shows the Variations according to Age in the rate of Mortality, of 3456 cases of Typhus Fever, admitted into the London Fever Hospital. (Compare with Diag: X.)

without taking into account the ages of the patients. In youth, it is far from being a fatal disease; but, in middle and advanced life, it is most mortal. These facts may be ascertained, by comparing the mean age of the fatal cases, with that of those which recover; or still better, by determining the rate of mortality in each period of life. Both plans have been adopted, with regard to the cases admitted into the London Fever Hospital, during ten years, and the results are embodied in Tables XIV. and XV, and in Diagrams II. and V.

TABLE XIV.

	Number	Mean Age.
Fatal cases in which age is known .	3456	29·33
Cases which recovered . . .	2753	26·15
Cases which died	703	41·78

Thus, the mean age of the cases which recovered being 26, that of the fatal cases was nearly 42 years. Moreover, this difference of age not only applied to the cases, admitted in the ten years collectively, but also held good for each of the individual years.

TABLE XV.

Age.	No. of Cases.	Deaths.	Mortality per cent.	Age.	No. of Cases.	Deaths.	Mortality per cent.
				Brought forward..	2832	391	
Under 5 years ...	17	3	17·65	From 45 to 50 yrs.	212	83	39·15
From 5 to 10 years	183	14	7·65	„ 50 to 55 „	150	78	52·
„ 10 to 15 „	363	18	4·95	„ 55 to 60 „	100	51	51·
„ 15 to 20 „	546	26	4·76	„ 60 to 65 „	88	49	55·68
„ 20 to 25 „	495	47	9·5	„ 65 to 70 „	42	28	66·66
„ 25 to 30 „	343	52	15·15	„ 70 to 75 „	24	17	70·83
„ 30 to 35 „	323	55	17·02	„ 75 to 80 „	6	5	83·33
„ 35 to 40 „	270	89	32·96	Above 80 years...	2	2	100·
„ 40 to 45 „	292	87	29·79	Age not known ..	50	11	22·
Carried forward	2832	391		Total	3506	715	20·39

From this Table, it appears that the rate of mortality is greater during the first, than during the second, ten years of life. Thus, the mortality during the first five years of life was upwards of 17 per cent.; in the second lustrum, it falls to 7·65; and between ten and twenty, it was under 5 per cent. After twenty, it went on progressively increasing, (see Diagram V.), until of those—

Above 30 years of age 36·05 per cent. died.
„ 40 „ 43·66 „
„ 50 „ 55·82 „
„ 60 „ 62·34 „

A curious circumstance is, that between forty and fifty-five, the mortality was considerably less, than in the period of life immediately preceding, and that this reduced rate of mortality was confined to females, being in them only 26·23 per cent. (48 deaths in 183 cases), while in males it was 35·7 per cent. (39 in 109).

The greater mortality of typhus fever in advanced life has been a matter of universal observation. Thus, of 363 cases of typhus admitted into the Edinburgh Infirmary in 1849, only 9 per cent. of those under twenty years of age died; but of those above thirty years, 40 per cent. died; and of those above fifty, one-half.[n] Of 361 cases under twenty, admitted into the same institution in the year ending September 30th, 1842, only 18, or 5·4 per cent. died; but of 253 above thirty, 70 (27·3 per cent.) died; and of 42 above fifty, 22, or more than one-half, died.[o] Similar observations were made by Dr. Steele in the Glasgow Infirmary in 1847, as follows:

	Cases.	Deaths.	Mortality per cent.
Under ten years. . . .	68	3	4·4
From 10 to 15 years . . .	195	10	5·1
„ 15 to 20 „ . . .	422	56	13·3
„ 20 to 30 „ . . .	942	176	18·6
„ 30 to 40 „ . . .	416	128	30·8
„ 40 to 50 „ . . .	256	91	35·5
Above 50 years . . .	100	46	46·[p]

Of 381 cases observed by Barrallier, the rate of mortality, according to age, was as follows:

	Cases.	Deaths.	Mortality per cent.
Under 30 years . .	381	90	23·6
Above 30 years . .	921	346	37·5
Above 50 years . .	156	92	59·[q]

4. *Station in Life.* An attempt has been made to calculate the influence of station in life, over the rate of mortality from typhus, by dividing the cases, admitted into the London Fever Hospital, into three classes, viz.: 1. Paying patients. 2. Free patients, unable to pay, but who have not been in the receipt of Parish relief, prior to their illness, and 3, Parochial paupers. The rate of mortality in each class, during 14 years, was as follows:

TABLE XVI.

	No. of Cases.	Deaths.	Mortality per Cent.
First Class - - -	94	14	14·89
Second „ - - -	2674	497	18·6
Third „ - - -	738	204	27·64

[n] *Statist. Tables*, 9th ser. p. 14.
[o] PEACOCK, 1843.
[p] STEELE, 1848, p. 161.
[q] BARRALLIER, 1861, pp. 281, 375.

The increased mortality in the third class was, to some extent, due to the more advanced age of the patients. Still, there is little doubt that, in London, the mortality from typhus is greatest in persons, whose constitutions have been weakened by previous want. On the other hand, it has been a common saying, especially in Ireland, that 'fever' is more fatal in the upper classes than in the lower.[r] This conclusion has been chiefly drawn from observations on clergymen and medical men, who, from their knowledge of the disease, have often great forebodings as to the result, which predispose to a fatal termination. The circumstance, however, is mainly due to the fact, that the cases of fever among the rich have been either typhus or enteric fever, whereas a large proportion of the cases among the poor, have been relapsing fever and febricula, which are rarely fatal. This is clearly shown by the Irish Reports of the epidemic of 1847.[s]

5. *Recent Residence in an infected Locality.* Of 2,941 patients affected with typhus, who had been resident in London more than six months, prior to their admission into the Fever Hospital, 532 or 18·09 per cent., died; whereas, of 160 patients, who had resided in London less than six months, only 18, or 11·25 per cent., died. This difference, however, was mainly, if not entirely, due to the greater age of the former class.

6. *Place of Birth and Race.* Dividing the patients with typhus admitted into the London Fever Hospital during fourteen years (1848—1861) into English, Irish, Scotch, and foreigners, the rate of mortality was as follows:

TABLE XVII.

	No. of Cases.	Deaths.	Mortality per cent.
English - - - - -	2756	500	18·14
Irish - - - - - -	350	52	14·85
Scotch - - - - -	28	5	17·85
Foreigners - - - -	33	12	36·36
Birth-place not noted -	513	194	38·6

The great mortality among 'foreigners' (who came from every part of the world), was not due to any difference of age. The mean age of the 12 fatal cases was only 37 years, whereas the mean age of all the fatal cases in the hospital was 42 years. In the Philadelphia epidemic of 1836, the mortality, according to Gerhard, was much greater among the blacks, than among the whites.[t] The great mortality among the patients, whose birth-place was not noted, is accounted for by the circumstance, that

[r] See BARKER and CHEYNE, 1821, i. 321, 329, 428, 467; BARTLETT, 1856, p. 256.
[s] *Irish Report*, 1848, vii. 103, 393, and viii. 84. [t] GERHARD, 1837, xix. 301.

most of them were so ill at the time of admission as to be unable to give any account of themselves.

7. Persons of a *feeble constitution* resist typhus less readily than the strong and robust.

8. It has been a matter of common observation, that *intemperate habits* greatly increase the fatality of typhus.

9. *Previous diseases* have a like effect. Hence, when typhus spreads in the wards of a general hospital, the mortality is often great.

10. *Mental depression* has also an unfavourable effect on the progress of typhus. This is no doubt one of the causes which renders typhus so fatal in prisons and besieged cities. Of 1,302 cases observed by Barrallier in the hulks of Toulon in 1855-56, 436, or more than one-third, perished.[u]

11. *Fatigue and Privation* before, and at the commencement of, the attack add greatly to the mortality. Persons, who waste their muscular power by struggling against the disease during the first few days, often become suddenly prostrate and die. During an epidemic, when it is difficult to find nurses for the sick, the immense amount of labour sometimes thrown on the devoted few who minister to their wants, not only predisposes them to be attacked, but renders the attack more fatal. The effects of fatigue, privation, and overcrowding in increasing the mortality, are also manifest, when typhus breaks out in armies in the field and in besieged cities. Of the French troops in the Crimea, one-half of those attacked died. According to Jacquot, of 12,000 cases of typhus among the French in the Crimea and at Constantinople during the first six months of 1856, 6,000 proved fatal. Among the Russians, even this rate of mortality was exceeded.[x] During the siege of Dantzick, it is stated that typhus carried off two-thirds of the garrison, and one-fourth of the population, numbers which indicate a frightful rate of mortality, as it is not probable that every individual was attacked.[y] Of 25,000 French troops, who escaped the disasters of the campaign of 1813, and who were afterwards besieged in Torgau, 13,448, or more than one-half, perished from typhus, within the space of four months.[z] Of the 60,000 troops composing the garrison of Mayence in 1813-14, there died of typhus 25,000.[a] Other instances, of an equally great mortality, have been collected by Gaultier de Claubry and Barrallier.[b]

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[u] Barrallier, 1861, pp. 281, 375.
[x] Jacquot, 1858, pp. 63, 150, 156. [y] De Claubry, 1838, ed. 1844, p. 41.
[z] Ib. p. 43. [a] Ib. p 45. [b] Barrallier, 1861, p. 120.

12. *Neglect of Treatment* increases the rate of mortality. In many patients, the good effects of removal from their overcrowded and badly ventilated dwellings, to the spacious wards of a hospital, are manifest in a few hours. In the Philadelphia epidemic of 1836, the mortality among the patients, under treatment from the commencement, was only 1 in 7, whereas it was 1 in 3, among those brought to hospital, late in the disease.[c] Dr. Mateer, from observations made at the Belfast Fever Hospital during seventeen years, ascertained, that the mortality from 'fever'[d] progressively increased, according to the duration of the illness before admission: of 1,625 cases admitted on the second or third day, only 54, or $3\frac{1}{3}$ per cent., died; of 5,921 cases admitted during the first week, 267, or $4\frac{1}{2}$ per cent., died; and of 3,667 cases, admitted during the second week, 397, or 10·8 per cent. died. Removal of the patient to hospital at an advanced stage of the disease, of itself, often adds to the danger. This was a point much insisted on by the late Dr. Alison[e]; and I have repeatedly known patients die from exhaustion, caused by their conveyance for several miles in a shaky vehicle.

c. Presence of certain Symptoms and Complications.

1. A presentiment of death, on the part of the patient, is very unfavourable. This is a common observation,[f] and I have had many opportunities of verifying it. It is most common in persons of the upper class, especially medical men.

2. It is a bad sign, if the pulse, in adults, exceeds 120, and especially if it be, at the same time, extremely soft and compressible, or small, fluttering, irregular, intermittent, reduplicate, or imperceptible. A sudden fall in the frequency of the pulse is always a favourable sign. On the other hand, typhus is occasionally fatal, when the pulse has never exceeded 100; and an unnaturally slow pulse points to serious impairment of the heart's action.

3. Complete absence of the cardiac impulse, and an inaudible systolic sound, are indicative of greater danger than any condition of the radial pulse.

4. A very excited, or thumping, action of the heart, associated with a very feeble radial pulse, is extremely unfavourable.

5. Hurried respirations, whether cerebral, or the result of pulmonary disease (see pages 137 and 182), are unfavourable.

6. Sleeplessness, especially when associated with delirium, and not yielding to treatment, is a very bad sign.

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[c] GERHARD, 1837, xx. 321. [d] MATEER, 1836; BARTLETT, 1856, p. 255.
[e] ALISON, 1844, p. 451, and *University Lect.* 1849 (not pub.) [f] LYONS, 1861, p. 194.

7. Speaking generally, the danger in any case may be measured by the severity of the cerebral symptoms, and is greater, the earlier these symptoms appear. The greater the headache, the more complete the loss of consciousness, the greater and the more constant the delirium, and the more profound the stupor, the greater is the danger.

8. The state of complete coma-vigil is invariably fatal.

9. Extreme contraction of the pupil is a bad indication. Dr. Graves regarded '*a pinhole pupil*' as an almost fatal sign.[h]

10. Deafness is not unfavourable, but neither is it a favourable symptom, as has been commonly believed (see page 170).

11. The danger is always great, in proportion to the degree of prostration. Extreme prostration, at an early stage, is always a bad sign. It is a favourable sign, when a patient, after lying for days on his back, helpless and motionless, turns round and sleeps on his side.

12. Muscular tremors, and still more carphology, subsultus, and spasmodic twitchings of the muscles of the face are of bad omen. Dr. Henderson found, at the Edinburgh Infirmary, in 1838 and 1839, that subsultus, to any considerable extent, was almost always followed by death.[i] Still, I have known many cases, where carphology and subsultus have existed for several days, and yet the patient has recovered.

13. Urgent and protracted hiccup usually terminates in death.

14. Permanent rigidity of the muscles of the limbs and strabismus are very bad signs.

15. Relaxation of the sphincters before the tenth day is a bad sign; after this, it is not uncommon in severe cases, which recover. Retention of urine is even more unfavourable than incontinence.

16. Extreme tympanitis, associated with symptoms of great nervous prostration, is always unfavourable.

17. A dry, brown, hard, retracted, tremulous tongue is a bad sign; but many cases, where the tongue presents these characters, recover.

18. The more abundant and the darker the eruption, *cæteris paribus*, the greater the severity and the danger of the case. The presence of numerous petechiæ, purpura-spots, or vibices, is particularly unfavourable. Cases without rash are usually mild and rarely fatal, except from complications.

19. Great lividity of the face and extremities, and a dusky erythematous condition of the skin, on the dependent parts of the body, are unfavourable.

[h] GRAVES, 1838. [i] HENDERSON, 1839.

20. Perspiration, in itself, is not a favourable, symptom, unless accompanied by other marks of amendment. Profuse and continued sweating, coldness of the surface, cold breath, and a rapid, weak pulse are almost fatal signs.

21. The prognosis is favourable, according to the freedom of excretion of urea and uric acid. Although a large amount of these products in the urine indicates great febrile action, it is better that they should be eliminated, than retained in the system. A sudden diminution in the amount of urea, while the fever progresses, is particularly unfavourable.

22. The presence of albumen or of blood in the urine, especially before the tenth day, or, if associated with casts of the uriniferous tubes, is also unfavourable, as indicating a condition of the kidneys, opposed to the free elimination of urea.

23. Albuminuria, or great diminution, or suppression, of urine, is often followed by convulsions and coma terminating in death.

24. The greater the number of unfavourable symptoms in one case, the greater is the danger.

25. The sudden cessation, at the end of the second week, of several of the unfavourable symptoms indicates the approach of convalescence. The first signs of amendment are a diminution in the rapidity, and an increased strength, of the pulse, and a slight return of appetite, while the tongue becomes clean and moist at the edges. By the experienced eye, a change can also be recognized in the patient's manner and countenance. The dusky tint of the face diminishes; the countenance is less stupid, and the conjunctivæ less injected, while, at the same time, the patient takes more notice, and answers more rationally.

26. The presence of any complication is always unfavourable.

27. Among the complications from which the greatest dangers may be apprehended, are pulmonary hypostasis and bronchitis (especially if stimulants, expectorants, and counter-irritation, fail to produce any good effect in a few hours), pneumonia, gangrene of the lung, convulsions, pyæmia, erysipelas, parotid-, and other inflammatory swellings, bed-sores, gangrene of the extremities and of the mouth, renal disease and scurvy. The gouty diathesis, from its being so often associated with disease of the kidneys, is a very serious complication. I have never known a gouty person, attacked with typhus, recover.

28. Even in the worst cases, the physician must never despair. Patients occasionally recover, whose deaths have for days appeared inevitable. In no disease, is this observation more commonly made, than in typhus.

d. Mode of Fatal Termination.

It is important to study the mode of fatal termination in typhus, both as regards prognosis and treatment. Death from the primary fever may take place by syncope or coma. In the one case, the heart's action is enfeebled from paralysis or disease of its muscular tissue; in the other, the blood undergoes modifications from the admixture of urea and other products of the retrograde metamorphosis of tissue, and from the diminution of red corpuscles, etc., which incapacitate it for supporting the changes, necessary for life. Most commonly, death is caused by a combination, in varying proportions, of syncope and coma. As a rule, from which there are few exceptions, the patient is quite unconscious for a considerable period prior to death. Lastly, in many cases, perhaps the larger number, death is due to one of the complications already mentioned, before, or after, the cessation of the primary fever.

Sect. XII. Anatomical Lesions.

THE most extensive results of *post-mortem* examinations of typhus yet published, are those of Messrs. Gerhard and Pennock[i] (50 cases); A. P. Stewart[k] (22 cases); John Reid[l] (147 cases); Thomas Peacock[m] (31 cases); William Jenner[n] (43 cases); Felix Jacquot[o] (41 cases); and Barrallier[p] (166 cases). My own observations, amounting to 54, entirely confirm the results arrived at by those authors, as do also the dissections of many hundreds of dead bodies, at the London Fever Hospital, during the last fourteen years.

Speaking generally, the appearances found after death from typhus are of a negative character. There is no constant or characteristic lesion. The chief abnormal appearances are here given.

a. Generalities.

1. *The Cadaveric Rigidity* is of short duration. Of 34 cases examined by Jenner, at varying intervals up to fifty-two hours after death, it was absent in 26, or 79·4 per cent., and was well marked in only 8.

2. *Emaciation.* Death often occurs, before there has been time for the body to emaciate.

3. *Putrefaction.* In many cases, there is a tendency to rapid

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[i] Gerhard, 1837. [k] Stewart, 1840. [l] Reid, 1840 and 1842. Eight of Reid's cases were examples of enteric fever. [m] Peacock, 1843. Three of Peacock's cases were enteric fever. [n] Jenner, 1849, No. 2.
[o] Jacquot, 1858. [p] Barrallier, 1861.

putrefaction after death, more rapid than after death from other diseases, at the same time of the year.

b. Integuments and Muscles.

1. *Discolorations.* In all cases, there is more or less livid discoloration, either general, or in patches, of the integuments on the dependent parts of the body. Occasionally, this lividity extends along the sides of the trunk, or even over the greater part of the body. The face is often livid.

In some cases, there is a green or dirty-purple discoloration of the skin, corresponding to the course of the large sub-cutaneous veins of the neck and extremities.

The walls of the abdomen and chest sometimes exhibit a green discoloration, within forty-eight hours after death. This is due to the action of some gas generated in the bowels, or in a gangrenous lung, for those parts of the skin protected from the action of such a gas, such as the skin over the liver, a distended bladder, or a rib, remain longest unchanged.

2. *The Eruption.* When death occurs before the cessation of the primary fever, and not from any subsequent complication, many of the darker spots constituting the eruption, are found to persist in the dead body. The microscopic characters have been already described. (See page 129.)

3. *Erysipelas.* (See page 197.)

4. *Bed-Sores and Gangrene.* (See page 198.)

5. *The Muscles* are often unusually dark. Jenner found this to be the case, in 6 out of 38 fatal cases. The muscular tissue also is often softer and more friable than natural; the frequency of this condition has been much insisted on by Stokes. Extravasations of blood are occasionally found in the substance of the muscles, in cases where no external violence has occurred. Jenner records two cases where hæmorrhage took place into the substance of the rectus abdominis muscle,[q] and Barrallier mentions a case, where there was extensive ecchymosis of all the abdominal muscles.[r]

c. Organs of Digestion.

1. *Pharynx* and *Œsophagus.* The lining membrane of the pharynx occasionally exhibits signs of recent inflammation. It is vividly injected, or of a dusky-red hue, and sometimes the mucous follicles are enlarged and contain a puriform fluid, or collections of puriform matter are found in the areolar tissue, behind the pharynx.

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[q] Jenner, 1850, xxi. p. 15.

[r] Barrallier, 1861, p. 279.

According to Jenner, the corpuscles in this matter have no nuclei like those of pus. The injected mucous membrane may be covered with viscid mucous or with diphtheritic flakes. The same appearances are occasionally found in the œsophagus. Recent ulceration is never found, either in the pharynx or œsophagus, after death from typhus. In 39 of 67 cases, observed by Jacquot and Barrallier, the pharynx was normal.

2. *The Stomach*, in a large proportion of cases, is perfectly healthy, and the only morbid changes, occasionally exhibited by it, are redness, mammillation, and softening of the mucous membrane. Of 78 cases, examined by Jenner and Jacquot, the mucous membrane of the stomach was pale and healthy in 46, or in 59 per cent. Of the remaining cases, there were patches of punctiform, or ramified injection, in 10, and minute ecchymoses in 5.

Of 75 cases, noted by the same observers, the mucous membrane was softened in 17, or in 22 per cent. This *ramollissement* was either general (7 cases), or limited to the great *cul de sac* (10). In 4 of Jenner's cases, there was such extreme softening of the great *cul de sac*, that it ruptured in the removal or washing of the organ. In a few instances, the consistence of the membrane is firmer than natural (7 of the 75 cases); but this condition is probably, in most cases, due to old disease.

Mammillation of the mucous membrane was noted by Jenner, in 7 out of 14 cases. In 1, it was general; in 6, it was limited to the vicinity of the pylorus. Mammillation of the mucous membrane of the stomach, towards its pyloric extremity, was frequently noted by Gerhard and Pennock.

Ulceration of the mucous membrane, as a consequence of typhus, is scarcely ever observed. I have never met with such an instance myself, and in none of Jacquot's and Barrallier's 207 cases is any mention made of such a lesion. In one only of Jenner's 43 cases, was any ulceration detected. 'Three inches 'from the pylorus, scattered over a space about an inch and 'a half in circumference, and seated on the posterior wall 'of the stomach, were nine ulcers, varying in size from a pin-point 'to a No. 4 shot; their edges were well defined, and not dis-'coloured.'[s]

3. *The Duodenum.* Of 75 cases, in which the duodenum was examined by Jenner and Jacquot, the mucous membrane was perfectly healthy in 60, or in 80 per cent. In the remainder, it

[s] Jenner, 1849 (2).

presented similar morbid appearances (various degrees of softening and injection), to those found in the stomach, and in most cases the stomach was similarly affected. In no case, have any traces of recent ulceration been discovered in the duodenum.

4. *The Jejunum* and *Ileum* exhibit no characteristic lesions; in most cases, the mucous membrane throughout is perfectly healthy.

Invaginations of the small intestines were found by Barrallier in 3 cases; but in none, was there any adhesion, or sign of inflammation, around the invaginated bowel.

In 34 out of 39, cases examined by Jenner, the colour of the mucous membrane was normal; in 2 cases there were hæmorrhagic spots beneath the mucous membrane, varying in size from a pin's head to a line and a half in diameter; in 1 case, the jejunum was injected, while the ileum was pale; in another, the fine injection was limited to the lower part of the ileum; and in the last case, both divisions of the bowel were of a deep-grey tint. Marked capillary injection was observed by Jacquot, in only 6 out of 41 cases. In my own cases, the presence of injection was exceptional; it was observed as often in the upper part of the intestines as in the lower, and it was never restricted to, or more intense in, the neighbourhood of Peyer's patches. Barrallier and other writers confirm Jenner's observations, as to the occasional occurrence of ecchymoses beneath the mucous membrane.[t]

Softening of the mucous membrane was observed in some cases by Reid, and occurred in 18 out of 78 cases (23 per cent.) collected by Jenner and Jacquot. In 13 of the 18 cases, the softening was general; in 5, it was partial.

The peculiar disease of Peyer's patches, and of the solitary glands, which constitutes the anatomical lesion of the so-called typhoid fever, is never found in exanthematic typhus. The evidence on this point is now overwhelming, although a few observers, who refuse to recognize any distinction between the symptoms of the two fevers during life, still publish cases of 'typhus' with intestinal disease.

Of 50 cases of typhus, examined by Messrs. Gerhard and Pennock, of Philadelphia, in 1836, 'the glands of Peyer were found 'not merely free from the peculiar lesions occurring in dothinen-'teritis or typhoid fever, but these follicles and the rest of the 'intestine, were more healthy in the petechial fever, than in the 'majority of other diseases. We are the more certain of the state 'of these glands, because our attention was closely directed to this

[t] Barrallier, 1861, pp. 109, 271.

'subject, and we had previously made most numerous examina-
'tions of the glands, in typhoid fever and in other diseases; we
'could, therefore, pronounce, with certainty, as to their actual
'condition. . . There was but in one case, *and that doubtful in its*
'*diagnosis*, the slightest deviation from the natural appearance of
'the glands of Peyer. In the case alluded to, in which there had
'been some diarrhœa, the agglomerated glands of the small in-
'testine were reddened and a little thickened; but there was no
'ulceration, and no thickening or deposit in the submucous tissue.
'The disease of the glands resembled that sometimes met with, in
'small-pox, scarlet fever, or measles, rather than the specific lesion
'of dothinenteritis. In all other cases, the glands of Peyer were re-
'markably healthy in this disease, as was the surrounding mucous
'membrane, which was much more free from vascular injection,
'than it is in cases of various diseases, not originally affecting the
'small intestine.'[u]

In 1840, similar results were published by Dr. A. P. Stewart. Out of a large number of cases of typhus, examined by him at Glasgow, in 1836, in not one did he discover any ulceration or evidence of the specific lesions of typhoid fever. In a few exceptional cases (2 out of 21), he found the patches distinctly elevated above the surface; but he pointed out that the appearance in question was not that, which characterizes typhoid fever.[x]

Of 43 autopsies of typhus cases made by Jenner, Peyer's patches were perfectly healthy in all but 3, *i. e.*, they were neither elevated, reddened, softened, nor ulcerated. Of the three exceptional cases, one was a case of tubercular ulceration; a second was an example of dysentery, in which the inflammation extended somewhat higher up the ileum than is usual, and involved the mucous membrane covering the elliptic patches, in common with that around them; in the third case, there was merely slight injection of one patch, but no ulceration.[y]

Dr. Peacock, who, as pathologist to the Edinburgh Royal Infirmary, and Physician to the Royal Free and St. Thomas's Hospital, has had unusual opportunities of examining the bodies of persons who have died of typhus, says, that 'Peyer's patches are
'usually less distinct, than in persons who die of other acute affec-
'tions, of similar duration.'[z]

Dr. Wilks' experience, at Guy's Hospital, has been precisely the same. In no fatal case of typhus has he found any disease of the small intestine.[a]

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[u] Gerhard and Pennock, 1837, xix. 302, and xx. 289.
[x] Stewart, 1840, p. 332. [y] Jenner, 1849 (2). [z] Peacock, 1856.
[a] Wilks, 1855 and 1856.

Jacquot, in his work on the Crimean typhus, has collected upwards of 400 cases, in not one of which were the lesions found after death, which characterize the *fièvre typhoïde* or *dothinentérite* of French writers. He observes: 'L'absence des lésions dothinen-'tériques dans le typhus de l'armée d'Orient est aujourd'hui une 'vérité acquise; il ne reste, à notre connaissance, qu'un médecin, 'qui soutienne le contraire, c'est M. Cazalas; mais, comme il con-'fesse, qu'il ne peut distinguer un typhus d'une fièvre typhoïde, 'son assertion n'a dès lors plus rien d'étrange.' Jacquot himself found ulceration of the mucous membrane in 5 out of 41 cases, but in all, the lesions were quite distinct from those of *dothinentérite*. In 2, the ulcerations appeared to have resulted from sloughing of the membrane, over a patch of submucous ecchymosis; and in 3, they were merely abrasions of the softened membrane; in none, was there any deposit in, or elevation of, Peyer's patches, or of the solitary glands, or any enlargement of the mesenteric glands.[b]

Equally conclusive evidence is borne by M. Barrallier, from his observations at the hulks of Toulon. He observes:—"Je ne jamais 'observé sur les 166 sujets nécropsiés pendant les deux épidémies 'du bagne, aucune des altérations des plaques de Peyer et des 'follicules de Brunner, que l'on rencontre dans la fièvre typhoïde.' To show the care with which he investigated the matter, he adds: '—Les intestins ont été toujours détachés du cadavre, incisés 'longitudinalement, étalés sur des planches disposées à cet effet, 'et étudiés soit à l'œil nu, soit à la loupe, et quelquefois sous l'eau; 'enfin rien n'a été oublié, pour pouvoir reconnaître et constater la 'moindre lésion.' All the dissections were made in public, by his colleague, M. Beau.[c]

In 1856, M. Godélier dissected 8 cases of typhus, which terminated fatally in the hospital of Val de Grace, and wrote as follows:—'Quand à l'altération caractéristique de la fièvre ty-'phoïde, des plaques de Peyer saillantes, molles ou dures, érodées 'ou ulcérées, et l'engorgement des ganglions mésentériques, nous 'ne l'avons jamais rencontrée.'[d]

Lastly, of 54 cases carefully examined by myself, in not one was there any deposit in, or ulceration of, Peyer's patches, at all resembling the appearances found after death from 'typhoid fever.' In 6 only of the cases, the glands were slightly more prominent than usual, but not more so, than is seen after death from many diseases; in 3 of the 6 cases, the glands presented the appearance compared by French pathologists, to a newly-shaven beard.

[b] JACQUOT, 1858, pp. 234, 256. [c] BARRALLIER, 1861, pp. 110, 265.
[d] GODÉLIER, 1856, p. 894.

This appearance was present in 4 of Jenner's 43 cases, in 8 of Jacquot's 41 cases, and in about one-third of Barrallier's 166 cases. It consists in patches of minute black dots, without any thickening or prominence of the mucous membrane. These patches are found in any part of the small intestine, and are often most numerous in the upper portion. They do not constitute part of the specific lesion of enteric fever, as has been imagined, for they are found after death from many other diseases, such as cholera, phthisis, etc.

The absence of any specific intestinal lesion in typhus will be again referred to, in discussing the points of distinction between this disease and pythogenic fever; but it may be here stated, that all the observers, whose experience has been referred to, have had ample opportunities, for studying the intestinal lesions of the latter disease.

5. *The Large Intestines* are usually quite healthy. In 28 of 37 fatal cases, examined by Jenner, and in 23 of Jacquot's 41 fatal cases, they exhibited no signs of recent disease.

At other times, the mucous membrane of this portion of the bowel is more or less injected; and now and then, there are indications of actual inflammation. Dysentery, in fact, in some epidemics, is a common complication of typhus. In 8 of Jacquot's 41 cases, in 4 of 37 cases noted by Jenner, in 5 of 132 cases examined by Reid, and in 3 of 54 cases examined by myself, signs of colitis were discovered, the membrane being bright-red, soft, and tumid, and covered with patches of lymph. In Reid's cases, the inflammation extended to the lower part of the small intestine, but there was no enlargement or ulceration of Peyer's patches. In 3 of Jacquot's cases, the inflammation of the colon had proceeded to ulceration.

It follows, that serious lesions of the bowels are occasionally found in typhus; but they are totally different from those, which characterize enteric fever.

6. *The Mesenteric Glands* are almost invariably healthy. In 2 of my cases, they were slightly enlarged, and of a dark livid hue, owing to extravasation beneath the enveloping peritoneum. Similar observations were made by Barrallier. In Gerhard's cases, they were always normal, or but very slightly injected. Of Jenner's 43 cases, they were healthy in 41 and contained tubercle in 2. Of 38 cases noted by Jacquot, they were slightly enlarged in 5 only, and in none did they contain deposit of morbid material.

7. *The Spleen*, in a considerable number of cases, is healthy (in 7 of 22 cases, Peacock; in 18 of 41 cases, Barrallier; and in two-

thirds of 166 cases, Barrallier). The chief abnormal appearances, presented by it, are hypertrophy and softening.

It was hypertrophied in two-thirds of the cases examined by myself; in one-half, examined by Jacquot; in one-third by Gerhard; and in scarcely one-sixth of those noted by Barrallier. The normal weight being between 4 and 5 ounces, the average weight in 34 cases of typhus was ascertained by Jenner to be 7oz. 5dr., and in 2 of the cases, it weighed as much as 14 ounces.

The consistence was diminished, in 15 of 22 cases examined by Peacock, and in 13 of 31 cases dissected by Jenner. Not unfrequently, the organ is reduced to a reddish-brown pulp, which runs out when the capsule is divided. Softening is more common after, than before, 50 years of age, and before, than after, the fourteenth day of the disease.

Jacquot mentions a case, where instant death resulted from rupture of the spleen.[e] In one instance, I have met with a patch of gangrene on the convex surface, of the size of a crown-piece, and extending the third of an inch into the substance. (See also p. 197).

8. *The Liver and Gall-Bladder.* The liver is occasionally healthy (in 16 of 41 cases, Jacquot; in 31 of 166 cases, Barrallier); but more commonly, it is hyperæmic, or its consistence is reduced. It was hyperæmic in 17 of 41 cases observed by Jacquot, in 7 of 36 cases observed by Jenner, and in 62 of 166 cases noted by Barrallier; its consistence was reduced, in 22 of Jenner's 36 cases, and in 40 of Barrallier's 166 cases. According to my experience, the liver is more commonly hyperæmic and tolerably firm, if death occurs on, or before, the twelfth day; but after this, it is often pale, flabby, and very friable. In every case, where I have subjected this softened hepatic tissue to microscopic examination, I have found an increased amount of oil in the secreting cells. Frerichs has found leucine, tyrosine, and hypoxanthine, in large quantity, in the liver of typhus and of other blood-diseases.

Messrs. Barudel and Jacquot met with a singular alteration of the liver, in four cases of typhus, which the latter observer designated *pulmonisation du foie.* 'Le parenchyme était d'un brun 'verdâtre livide, criblé de variles, aérolaire, spongieux, mou, 'friable, évidemment crépitant, contenant un peu de liquide 'spumeux, mêlé de bulles de gaz.'[f] These were evidently examples of that rare lesion, described by Frerichs, as 'Emphysema 'of the Liver,' and believed by him, to be due to a process of local disintegration.[g]

[e] Jacquot, 1858, p. 235. [f] Ib. p. 250.
[g] *Diseases of Liver*, Syd. Soc. Transl. ii. 370.

There is never any ulceration of the lining membrane of the gall-bladder. The bile is usually dark-green, or greenish-yellow, and of ordinary consistence.

9. *The Pancreas,* like the liver, is frequently found to be hyperæmic, when death occurs at an early stage; at a later stage, its consistence is often reduced. In the epidemic at Toulon, Barrallier found the pancreas, in most cases, hyperæmic and slightly hypertrophied.

10. *Peritoneum.* With the rare exceptions already mentioned, signs of recent peritonitis are never found after death from typhus. A small quantity of *post-mortem* serous effusion is occasionally found, and now and then small ecchymoses are observed in the sub-peritoneal tissue. (See page 196.)

d. Organs of Circulation and Blood.

1. *The Pericardium* often contains an increased amount of serosity, which occasionally presents a deep-red tint, owing to the transudation of the hæmatine of the blood. When this is the case, the fluid contains no blood-corpuscles, and the posterior surface of the heart presents patches of dusky-red staining, or ecchymoses. In one case, Jacquot observed signs of recent pericarditis.[h]

2. *The Heart.* In a large number of cases, the muscular tissue of the heart is flabby, soft, and easily torn. These characters were noted by Peacock, in 7 of 19 cases; by Jenner, in 15 of 29 cases; by Jacquot, in 7 of 39 cases; and in about one-third of my cases; they are rarely absent, when the circulation has been extremely feeble for some days prior to death, and when the fatal event has been due to syncope rather than to coma. The softening is independent of the duration of the disease, the age of the patient, the external temperature, or the interval since death. In many cases, it is confined to the left side of the heart. (See page 136.)

Lænnec was the first to describe softening of the heart, as a consequence of idiopathic fever. According to him, it was always most marked, when putrid (typhoid) symptoms had been most prominent, and it was merely part of a general softening of the muscular system.[i] Some years later, Louis described softening of the heart, as a common lesion in 'typhoid fever';[k] and in 1839, Dr. Stokes recorded a number of cases of both typhus and 'typhoid fever,' to shew the importance of this condition, as accounting for certain cardiac phenomena during life, already referred to.[l]

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[h] Jacquot, 1858, p.230. [i] *Traité de l'Auscult. Méd.* 2me ed. 1826, ii.537.
[k] Louis, 1829 (ed. 1841, i.298). [l] Stokes, 1839; also work on *Diseases of the Heart,* p.371.

Rokitansky,[m] and other pathologists, have stated, that this softening is 'a simple diminution of consistence, not depending upon any 'disturbance of texture.' But, of six cases, where I subjected the heart, in this state, to microscopic examination, in every one, there was distinct fatty degeneration of the muscular tissue; the transverse striæ were at many places indistinct or absent; and the fibrils contained numerous granules or minute oil globules. Similar appearances have been found by Dr. Joseph Bell, in five cases of Continued Fever, several of the patients being of an age, at which fatty degeneration could scarcely have been expected, as an independent lesion.[n] Dr. Bell believes that the appearances found by him were due to inflammation, and refers to Virchow's statement, that myo-carditis may give rise to fatty degeneration.[o] But if the term *inflammation* implies exudation of fibrine, then the myo-carditis of Virchow is not inflammation; for, according to Virchow himself, no fibrinous exudation is to be found in ordinary myo-carditis.[p] Although the propriety of using the term *inflammation*, in such cases, must depend on the meaning attached to it, it seems to me more correct, to regard these cases, as examples of acute fatty degeneration, due to a paralysed condition of the sympathetic, and to the consequent increased disintegration, and diminished nutrition, of the muscular system.

3. *Endocardium.* The lining membrane of the heart and of the great vessels is often observed, stained of a dusky-red (in 12 of 24 cases, Jenner; in 6 of 41 cases, Jacquot). Both sides of the heart may be thus affected, but the right, more commonly than the left. Although this staining is of a *post-mortem* nature, it indicates a great alteration of the blood. It is usually associated with softening of the muscular tissue.

Signs of recent endocarditis are extremely rare. One case is mentioned by Jacquot, and another occurred recently at the London Fever Hospital.

4. *The Blood* undergoes remarkable changes in typhus. In the first place, it is darker and more fluid than natural. Sometimes the blood, found in the heart and great vessels, is perfectly liquid, without any trace of clot; at other times there are a few soft, black clots, mixed with dark fluid blood. These characters were found by Reid in 28 of 61 cases; by Peacock, in 14 of 21 cases; by Jenner, in 17 of 37 cases, and by Jacquot in 18 of 41 cases. When pale coagula are found, they are usually soft and friable,

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[m] *Path. Anat.* Syd. Soc. Transl. iv. 171. [n] BELL, 1860.
[o] *Cellular Pathology*, Dr. CHANCE's Transl. p. 352. [p] Ibid. p. 390.

and mixed with dark blood. Firm, pale, fibrinous clots are very rare (in 2 of 61 cases, Reid; in 4 of 37 cases, Jenner), and are chiefly observed in cases, where death has resulted from some complication, after the cessation of the primary fever. The blood, taken from the body during life, often coagulates imperfectly, the crassamentum being soft and diffluent, and rarely exhibiting the buffy coat. Typhus blood is more apt to become putrid than healthy blood, or than the blood, in other diseases. On more careful examination, there is found to be a marked diminution of fibrine, as well as of the other solid constituents; the red corpuscles are diminished, although increased relatively to the amount of fibrine.[q] These changes are most obvious in the latter stages of the disease, and in those cases, where typhoid or putrid symptoms have been most marked. Researches are still wanting on the changes in the saline constituents of the blood in typhus, more particularly in reference to the non-appearance of chlorides in the urine. According to the observations of Lehmann,[r] the salts are increased, rather than diminished, as was formerly thought. When the blood is very fluid, the red corpuscles are found, under the microscope, to be crenate and mis-shapen, as if undergoing solution, and they are loosely aggregated in amorphous heaps, in place of adhering in rolls. Whether the natural fluid condition of the blood during life be due to the presence of ammonia,[s] or to the vital action of the neighbouring tissues, there is good reason for believing, that the unnatural fluid state, in typhus, results from an abnormal amount of ammonia, possibly derived from the decomposition of urea. At all events, blood, when artificially mixed with ammonia, presents the same appearances, to the naked eye and under the microscope, as in typhus; while, at the same time, there is evidence, as before stated, that the blood of typhus contains an increased amount of ammonia (see pp. 115 and 139).

e. Organs of Respiration.

1. *The Pituitary Membrane* not unfrequently exhibits a bright-red, or livid, hue.

2. *Larynx and Trachea.* Recent disease of the larynx is occasionally met with (in 6 of 26 cases, by Jenner; in 16 of 39 cases, by Jacquot). The lining membrane is of a bright-, or dusky-red hue, tumid and covered with viscid mucus, or with diphtheritic flakes, or with a puriform fluid; its texture is softened, and sometimes the mucous follicles are enlarged. Jacquot observed diphtheritic

[q] Carpenter's *Pr. of Hum. Phys.* (5th ed.) p. 175.
[r] Lehmann's *Phys. Chemistry*, Day's Transl. ii. 262, 266. [s] Richardson, 1858.

exudation, in 2 out of 39 cases. In some instances, œdema glottidis is found. Dr. Buck has published coloured plates of œdema glottidis, occurring in the typhus of Irish immigrants to America.[t] It is only in exceptional cases, that the larynx is ulcerated (1 in 26, Jenner; 4 in 39, Jacquot; and 1 in 166, Barrallier); and then the ulcers are always minute and superficial. These morbid appearances in the larynx are almost always accompanied by signs of inflammation in the pharynx.

3. *Bronchi.* Signs of catarrhal inflammation are among the most common *post-mortem* appearances in typhus. The lining membrane is of a bright-, or dusky-red tint, and more or less filled with tenacious frothy secretion. These appearances were present in 18 of 20 cases observed by Peacock, in 20 of 22 cases observed by Jenner, and in 19 of 41 cases observed by Jacquot.

4. *The Lungs* are rarely healthy. Of 146 cases, examined by John Reid, Peacock, Jenner, and Jacquot, they exhibited some deviation from health, in all but 6.

The most common morbid appearance is hypostatic congestion. In a slight form, this condition is rarely absent; and it is certainly far more common than after death from other diseases in which the lungs are not primarily affected, while in not a few cases (in 21 of 131, Reid; in 11 of 35, Jenner), the affection amounts to complete consolidation, so that the pulmonary tissue sinks in water, and does not crepitate. This consolidation is commonly mistaken for pneumonia, but is distinguished by the following characters. It is limited to, or greatest at, the most dependent parts of the lungs (which are not at the bases, but in the hollows of the fourth, fifth, and sixth ribs); from the posterior surface, the consolidation extends from one to three inches into the substance of the lung, and is not bounded by any defined margin, but passes imperceptibly into the surrounding crepitant tissue; its cut surface is smooth and non-granular, and of a dark-purple or chocolate colour, and exudes a quantity of non-aerated claret-coloured serum. Both lungs are usually affected in about an equal degree; but sometimes one lung is more implicated than the other, or the affection is limited to one organ.

Œdema of the lungs is sometimes the chief lesion, and is often greatest in the upper lobes, from which a large quantity of colourless serosity can be squeezed, as from a sponge. Œdema may be associated with pulmonary hypostasis.

True pneumonia is not a common lesion in typhus. It was present in 12 of 131 cases (Reid), in 9 of 35 cases (Jenner), in 2

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[t] Buck, 1848.

of 27 cases (Peacock), in 12 of 41 cases (Jacquot), and in 8 of 54 cases (Anderson). It may be lobular or lobar, but more commonly, lobular.

When pneumonia does occur, it occasionally terminates in gangrene, which exhibits the ordinary appearances of this lesion. Cases of this nature have been observed by Peacock, Jenner, and Barrallier, and several have come under my own notice.

5. *The Pleuræ.* Signs of recent pleurisy are rare after death from typhus (2 of 131 cases, Reid; 2 of 36 cases, Jenner; 5 of 41 cases, Jacquot). The effusion is usually fluid, and rarely assumes the form of plastic lymph. Simple serous effusion is occasionally met with (in 8 of 41 cases, Jacquot); and in rare cases patches of sub-pleural ecchymosis are observed.

f. Nervous System.

1. *The Cerebral Membranes* often exhibit increased vascularity, but never any deposit of lymph or pus, indicative of recent inflammation.

Of 24 cases examined by Peacock, there was increased vascularity of the pia mater in only 8.

In 10 out of 36 cases examined by Jenner, the dura mater was congested; in 22 of the 36 cases there was increased vascularity of the pia mater, the injection being trifling in 7, and intense in 7; in 13 cases, there was no increased vascularity.

Of Jacquot's 41 cases, the venous sinuses were found gorged with blood in 29; in 12, there was no engorgement. In 17 cases there was marked injection of the large veins of the meninges, and in 9 there was intense fine injection; but, in 13 cases, the injection was insignificant, or there was none at all.

The choroid plexuses are occasionally very vascular.

The increased vascularity of the cerebral membranes in typhus must not be regarded as a sign of inflammation, and does not account for the cerebral symptoms, observed during life. The vascularity is not greater, or more common, than when death results from disease of the lungs; and, in the majority of cases, where there is increased vascularity of the cerebral membranes in typhus, some impediment will be found in the pulmonary circulation, or there has been evidence of greatly impaired cardiac action. The congestion, in fact, is mechanical or passive, never active. Moreover, I am satisfied, from many observations, that there is no relation between the vascularity of the membranes and the symptoms. I have repeatedly known the most severe cerebral symptoms during life, without abnormal vascularity of the cerebral membranes after death. Although it has been stated, that inflammation of the

cerebral membranes occurs in typhus, I have never met with an instance, where the appearances justified such a conclusion, and this result accords with the experience of Reid, Peacock, Jenner, Jacquot, Barrallier, and most observers. M. Moering, of the Russian army, examined the cerebral membranes and sub-arachnoid serosity microscopically in upwards of 200 cases, but in no instance could he detect a single pus, or exudation, corpuscle.[u]

Hæmorrhage into the cavity of the arachnoid is a lesion in typhus, to which attention was drawn by Peacock (1 in 24 cases), in 1843, and which was found by Jenner in 5 out of 39 cases. In every case, the coagulum was in the form of a delicate film, varying in thickness, and, consequently, in hue, in different cases, and in different parts of the same clot. It is usually situated on the convex surface of the brain, and may extend over an entire hemisphere, or even to the base. In none of the cases, has the source of hæmorrhage been discovered; the brain has appeared healthy, and there has been no intense injection of the membranes. In one of Jenner's cases, blood was also extravasated into the substance of the rectus abdominis muscle. I have only met with this lesion once in upwards of 30 cases, where I have examined the head; and, in this case, the cerebral symptoms were comparatively slight. John Reid does not appear to have met with it once in 125 cases. Barrallier found it in only 1 of 166 cases. M. Moering found it in several cases in the Crimea.[x]

It is usually found, that the membranes can be torn from the brain with unusual facility, without removing any of the cerebral substance. Jenner noted this condition in 9 out of 11 cases. It occurs after death from many diseases, but it is certainly unusually common in typhus.

The Pacchionian bodies were noted by several observers in the Crimea, as increased in number and size (in 17 of 41 cases, Jacquot); but, so far as we know, such appearances have no pathological signification.

2. *The Sub-arachnoid Serosity and Ventricular Fluid.* Increased effusion of serum within the cranium is one of the most frequent morbid appearances in typhus. The most common seats of this effusion are beneath the arachnoid and in the lateral ventricles, and sometimes in the cavity of the arachnoid. The serum is always transparent and usually colourless. Sometimes it is straw-coloured; and occasionally it appears opalescent, owing to slight opacity of the superposed membrane. It never contains any flakes

[u] Jacquot, 1858, p. 253. [x] Ibid, p. 244.

of lymph or exudation-corpuscles. The quantity beneath the arachnoid may be enough to separate the convolutions, but is rarely sufficient to elevate the arachnoid; the amount in the lateral ventricles rarely exceeds two drachms, and that at the base of the cranium, in the cavity of the arachnoid, is seldom more than one fluid ounce.

Of 125 cases, in which the brain was examined by Dr. John Reid, the sulci were more or less wide and full of serum in 60; and in 25, the quantity was sufficient to elevate the arachnoid above the surface of the convolutions. Of 82 cases, in which the fluid in the lateral ventricles was carefully measured, in 37, it was less than half a drachm; in 37, it exceeded one drachm; in 23, it exceeded two drachms; and in 4, it varied from five drachms to an ounce and a-half.[y]

Of 23 cases, examined by Peacock, the sub-arachnoid serosity was scanty or absent in 15, of moderate quantity in 6, and so copious as to elevate the membrane above the surface of the convolutions in 2. The fluid in the lateral ventricles was more than half a drachm in 17 cases, half an ounce or upwards in 4 cases, and two ounces in 1 case.[z]

Of Jenner's 36 cases, more or less sub-arachnoid serosity was found in 23; in 25, serum was found in the cavity of the arachnoid varying in quantity from two drachms to two fluid ounces; the average amount of fluid in the lateral ventricles was two or three drachms.[a]

Of Jacquot's 41 cases, the sub-arachnoid serosity was trifling in amount in 20; in 16, it was abnormally abundant, and in 5, there was none at all. In 24 cases, there was no serosity in the cavity of the arachnoid; in 9, the quantity was considerable or abundant; and in 8 cases, there was an increased amount of fluid in the lateral ventricles.[b]

Barrallier met with an increased quantity of fluid in the ventricles in 30 of 138 cases; and occasionally with effusion of limpid fluid beneath the arachnoid.[c]

The increased amount of serosity within the cranium is no sign of inflammatory action, and accounts, in no way, for the cerebral symptoms during life. There is no relation between the severity of the cerebral symptoms and the amount of fluid. More than twenty years ago, it was shown by Dr. John Reid, as the result of an examination of the brain in 125 cases of typhus, that the

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[y] Reid, 1840 and 1842. [z] Peacock, 1843. [a] Jenner, 1849 (2)
[b] Jacquot, 1858, p. 226. [c] Barrallier, 1861, p. 267.

cerebral derangement was as strongly marked in those cases, where no increased effusion within the cranium was found after death, as in those, where the amount was excessive, and that occasionally there was very little cerebral derangement where the quantity was great. About the same time, Dr. Peacock arrived at the same results, and the fact is now admitted by most modern pathologists. If the reader has any doubt on the point, it will be at once removed, by referring to Dr. Reid's masterly exposition of the subject.[d] The quantity of fluid present within the cranium in typhus is not greater than is usually found in persons of an advanced age, or who have died from chronic emaciating diseases. Under such circumstances, as well as in typhus, the brain shrinks from want of proper nutrition, and the fluid is effused to fill up space. It does not exercise more than the normal pressure on the brain, and, as above stated, it does not account for the comatose symptoms of typhus.[e]

3. *The Cerebrum and Cerebellum* are often perfectly healthy; and the chief abnormal appearances which they present are, increased vascularity, indicated by an unusual number of bloody points on section of the white matter, a darker tint of the grey substance, and diminished consistence.

According to Reid, the vascularity of the brain-substance was increased in 34 of 82 cases; according to Jenner, in 15 of 36 cases; according to Peacock, in 6 of 24 cases, and, according to Jacquot, in 16 of 41 cases, altogether in 71 of 183 cases, or in 38·8 per cent. This increased vascularity, like that of the membranes, is no sign of inflammation, and has no relation to the cerebral symptoms. In fact, according to my experience, it is less common in typhus, than after death from some other diseases, such as affections of the lungs, where there has been no suspicion of cerebral disease; while, in some cases of typhus, where cerebral symptoms have been most strongly developed, I have found no increase of vascularity, or decided anæmia of the brain-substance. The increased vascularity of the brain-substance, when present, is, like that of the membranes, either mechanical or passive, never active. Of 12 cases examined by Peacock, where the brain or membranes were abnormally vascular, the lungs were diseased in all.

Softening of the brain has been observed occasionally by Reid, Jenner, Jacquot, Barrallier, etc. Jenner found the brain of normal consistence in 29, and more or less softened in 7, of 36

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[d] Reid, 1840 and 1842. [e] See Todd, 1860, p. 159.

cases. Of Jacquot's 41 cases, the consistence was normal in 27; there was softening in 12; and induration of both hemispheres, in 2 cases. Barrallier met with softening, in only 5 of 138 cases. The softening is either general or partial; and, in the latter case, it may affect the upper surface of the hemispheres, the inner surfaces of the optic thalami, the fornix, or corpus callosum. So far as my observation extends, this softening is always either cadaveric, owing to the length of time intervening between death and the inspection, or to the state of the weather, or it is produced by infiltration of serum from the neighbouring cavities. I know of no instance, where true softening, distinguished by the presence of compound granular corpuscles, oil globules, and disintegrated nerve tissue, can fairly be regarded as a result of typhus. According to Rokitansky, 'slight condensation of the brain is the rule in ' typhus, while decided softening, which, in fact, is nothing more ' than *œdema* of the brain, is certainly common, late in the disease.' [f]

Barrallier[g] has called attention to the remarkable indistinctness of the *arbor vitæ* of the cerebellum, in some cases. Of 28 autopsies, made by him during the epidemic at Toulon, in 1856, this phenomenon was observed in 7; and, in 2 of the cases, the *arbor vitæ* was almost completely effaced.

4. *The Spinal Cord.* Increased vascularity of the spinal membranes is less common, than in the case of the cerebral membranes. In most cases, the spinal fluid is somewhat increased. Softening, like that of the cerebral substance, has been occasionally noticed by Landouzy, Godélier,[h] and Jacquot.[i]

5. *The Sympathetic System* has not yet been examined with requisite care. M. Marmy found many of the ganglia softened, especially those of the neck.[k]

g. Urinary Organs.

1. *The Kidneys.* When it is considered, that chronic renal disease is found in fully one-fourth of the patients dying in a general hospital, it will not be surprising that it is not uncommon after death from typhus. But, in many cases of typhus, the kidneys exhibit unmistakeable evidence of recent disease, which varies in its character, according to the date of death. If death occurs before the fourteenth day, the organs are usually hyperæmic and hypertrophied, while the tubes are gorged with epithelium, and sometimes contain blood. I have known the kidneys present the

[f] *Path. Anat.* Syd. Soc. Transl. iii. 425. [g] BARRALLIER, 1861, p. 372.
[h] BARRALLIER, 1861, p. 106. [i] JACQUOT, 1858, p. 228. [k] Ibid.

appearances of acute nephritis, as intensely developed as in any case of scarlatina (See Case X). If death has occurred at a later stage, I have usually found the kidneys large and pale, weighing, perhaps, six ounces each, the outer surface smooth, the cortical substance hypertrophied and soft, and the tubes loaded with epithelium cells, containing an unusual amount of minute granules.

2. *The Bladder.* The mucous membrane is occasionally slightly injected, or marked by hæmorrhagic spots.

h. Genital Organs.

The genital organs, both in the male and female, never present any abnormal appearance attributable to typhus.

The *post-mortem* appearances of typhus may be summed up as follows:—

1. There is no lesion constant in, or peculiar to, typhus. The chief morbid appearances are, a fluid condition of the blood; hyperæmia of the cerebral membranes, and increased intra-cranial fluid; bronchial catarrh and pulmonary hypostasis; softening of the heart, liver, spleen, and pancreas; hyperæmia and hypertrophy of the kidneys. The relative frequency of these lesions varies, at different times and places; none are of constant occurrence, or peculiar to typhus.

2. The intestines never exhibit the peculiar lesions constantly present in enteric fever, and the mesenteric glands are not enlarged.

3. No evidence of recent inflammation is ever found in the brain or its membranes, to account for the cerebral symptoms.

Sect. XIII. Treatment.

THE treatment of typhus is divisible into prophylactic, and curative; the former consisting in the removal of those causes which are known to favour its origin and propagation; and the latter, in the application to individual cases of the resources of pharmacy and hygiene.

A. *Prophylactic Treatment.*

It is easier to prevent typhus than to cure it. Indeed, the means for preventing its origin are, in a great measure, within our power. The remarks already made on etiology have anticipated much that might be written on prophylaxis. To know the cause of a disease, is to know how to remove it.

The subject of prophylaxis resolves itself into two divisions;—

how to prevent the generation of the typhus-poison; and how to arrest its propagation.

1. *Rules for Preventing the Generation of Typhus-Poison.*

The one thing essential to the development of typhus, is overcrowding of human beings, with deficient ventilation, aided by whatever tends to debilitate the constitution. Remove the essential cause, and typhus will cease to exist. A century ago, there were no greater hotbeds of typhus than the jails of England; but, thanks to the philanthropy of Howard, the nation is now freed from such an imputation. What is wanted, is another Howard, to effect similar reforms in the dwellings of our poor, and in the accommodation of our soldiers and sailors; there can be little doubt, that the results would be equally successful. Although much has been done of late years by sanitary reformers, much remains to be effected.

It is difficult to fix the precise number of cubic feet required for each individual in a room. It has been calculated that an adult man expires about 160 cubic feet of air in twelve hours, containing about 4 per cent. of carbonic acid; but as air containing more than 1 per cent. of carbonic acid cannot be breathed without injury, it follows that a man, confined in an air-tight chamber for twelve hours, would require 640 (160 × 4) cubic feet of space, and double that space at the end of twenty-four hours. This is on the supposition that there is no ventilation; but the amount of space must always be in proportion to the amount of ventilation; and, in fact, cubic space is of far less importance than ventilation. A man shut up in an air-tight room will as certainly be poisoned, if the room be large, as if it be small; the only difference will be in the time required. The ventilation of a room, then, must be the basis of a true judgment. The amount of ventilation requisite to prevent a room from containing more than 1 per cent. of carbonic acid, is about 1½ cubic feet of air per minute, for each person.[1] Some authorities, such as Drs. Arnott and Reid, consider even this percentage of carbonic acid too great, and recommend as much as 10 or 20 cubic feet of ventilation per minute. The means for ventilation are either *constant* or *occasional.* The constant (the chimney and other unclosed openings) are more important than the occasional (doors and windows), and should be proportioned to the number of inmates. Indeed, the excessive use of occasional means

[1] For much valuable information on this subject, the reader is referred to a Report, presented to the Poor Law Board in 1856, by Dr. Bence Jones.

of ventilation is the best proof, that those in constant use are insufficient. If the air in a room contain more than 1 per cent. of carbonic acid, or be in the slightest degree fusty, it may be held that the ventilation is defective, or that the number of inmates is too great. Although our present ignorance obliges us to take the amount of carbonic acid, as the safest index of all the injurious substances which render ventilation necessary, this is not the only substance contained in air contaminated by over-crowding. Pure carbonic acid has no unpleasant smell or taste; whereas, the disagreeable fusty odour, produced by over-crowding with bad ventilation, is familiar to all.

From what has been stated, it may be inferred, that 500 cubic feet of space, with 2 cubic feet of ventilation per minute, constitute the smallest amount that can be safely allotted to each person.

The present regulations on the matter in London, are as follows: In workhouses, the amount of space, enforced by the Poor Law Board, is 300 cubic feet for a sick ward, or for a dormitory occupied by night only, and 500 cubic feet in a ward occupied both day and night.[m] In some districts of London, the vestry considers a house to be over-crowded, if the cubic space available for each individual falls short of 400 cubic feet.[n] The common lodging-houses of London are under the supervision of the police, who have the power to enforce an allowance of 250 cubic feet for each person. But, notwithstanding these regulations, which err, I think, in fixing too low a limit, and what is far more important, in not providing for proper ventilation, I have repeatedly known whole families living and sleeping in rooms, with not more than 120 or 150 cubic feet of space for each person, and this, with little or no ventilation. Such occurrences are particularly common in seasons of scarcity, or when large bodies of men are thrown out of employment. In either case, the poor flock from the country to the large towns, where the channels of charity are most numerous; and there swell the population of the already crowded lodging-houses and workhouses (See pages 49 and 53). It is at such seasons, therefore, that the authorities should be most on their guard against the known effects of over-crowding.

The prevention of scarcity of food, loss of employment, and other causes of destitution, is not always within human power; but, under such circumstances, every means, both public and

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[m] *Instructions of Poor Law Board to Boards of Guardians.*
[n] *Vestry Regulations of St. Giles' District.*

private, calculated to alleviate the distresses of the poor, should be adopted. Moreover, no time is to be lost in affording relief; it is difficult to stay the plague, when once it has begun. Care also must be taken, that the funds collected for such purposes, do not produce the very evils they are intended to avert. The poor naturally flock in greater numbers to those localities where most relief is to be obtained, and the result has often been increased crowding. The expediency of supplying relief to the poor, in their crowded dwellings, may therefore be questioned. A preferable plan would be, to establish, during seasons of scarcity, and when typhus is prevalent, temporary buildings of wood or iron, or tents, in the neighbourhood of large towns. Here, over-crowding could be prevented, the poor could be supplied with abundance of fresh air and food, while the number of persons resorting thither for relief would prevent over-crowding in the towns. The expense of such a plan would certainly not exceed, what the spread of an epidemic always entails.

Especial care must be taken to prevent over-crowding and bad ventilation, during winter; for although fires and the external cold increase the rapidity of the circulation of air, so that the openings for *constant* ventilation may be smaller, yet the poor are in the habit of closing every crevice to keep out the cold, and rarely resort to any means for *occasional* ventilation.

The dwellings of the poor ought to be so constructed, as to ensure good ventilation. Closed courts surrounded by high houses are always objectionable. Every window-frame ought to be moveable, and every room should be provided with means for *constant* ventilation. Human beings ought to be prohibited from living in underground cellars, where proper ventilation is impossible.

Common lodging-houses, and indeed every house in populous localities, should be thoroughly cleaned, and the walls lime-washed, twice every year, and oftener, when there is reason to apprehend an epidemic of typhus.

Inasmuch as squalor aggravates the evils of over-crowding, personal cleanliness should be encouraged among the poor, by the erection of free public baths, and wash-houses for their clothes.

Most of these remarks apply equally to workhouses, jails, transport- and emigrant-ships, barracks, and camps. *Typhus fever, which, during warfare, often commits greater havoc than the sword of the enemy, may be prevented by plenty of fresh air, and by personal cleanliness.* The regulations to be adopted must vary according to circumstances, but the general principles will always

be the same: no over-crowding, good ventilation, personal cleanliness, and a nutritious diet.

2. *Rules for preventing the Propagation of the Typhus-Poison.*

An abundant supply of fresh air is not only the best means for preventing the generation of typhus, but is the surest safeguard against its propagation to the attendants on the sick and to other persons. The truth of this statement has been already so fully established, that it is needless to enlarge upon it. But, as this desideratum is not always attainable in the houses of the poor, the infected persons ought to be isolated, and, if possible, removed at once to a hospital.[o] At the same time, the house should undergo a thorough cleansing and ventilation, the inhabitants should be reduced in number, their clothes washed, and every means taken, to ensure personal cleanliness.

When typhus is prevalent, no person, whether ill or not, ought to be admitted among the other inmates of a workhouse, without having a warm bath and other clothing, while his own clothes are being purified.

There cannot be a more reprehensible custom, than that of bringing patients labouring under contagious fevers to hospitals, in common street-cabs. Apart from the danger of the disease being thus propagated, the fatigue and shaking often inflict injuries on the patient, from which he never recovers. Scarcely a year passes, that several patients are not brought to the London Fever Hospital in cabs, who die either during the journey, or soon after admission. Fever patients ought always to be conveyed in covered litters, or spring invalid-carriages, constructed for the purpose, and maintained by the parochial authorities.

When a typhus patient is brought to a hospital, care should be taken to disinfect his clothes, before they are restored to him or to his friends. The under-clothing ought to be immediately immersed in water containing Condy's Fluid, or a small quantity of hydrochloric acid (ʒss. ad Oj.), and after twenty-four hours, washed, boiled, and hung out to dry in the open air. The outer clothing ought to be exposed, for some hours, to a dry heat of 212° Fahr., then subjected to the fumes of sulphurous acid or chlorine, and afterwards hung out in the open air, or in thoroughly ventilated wooden sheds, until the patient's recovery. Of all these measures, free exposure to the air is the most important.

[o] By *14th and 15th Vict.*, cap. 28, the keeper of a common lodging house is bound to inform the local authority immediately, when any inmate of such house is attacked by contagious disease; and by *16th and 17th Vict.*, cap. 41, the local authority has the power of removing such person to a hospital, on the certificate of the medical officer of the parish, that the disease is infectious.

The linen and bed-clothes, used by typhus patients, ought to be treated in the same way as the under-clothing worn before he takes to bed. In general hospitals, they ought to be kept separate from the clothes of other patients.

Every typhus patient, on admission into hospital, ought to have a tepid bath; or, if he be too weak, the body should be sponged all over with soap and water.

In hospitals, where typhus patients are admitted, there ought to be an allowance of at least 1500 cubic feet to each bed; the beds ought to be six feet distant from one another, and the freest ventilation should be maintained. Doors, windows, and other occasional means of ventilation must not be trusted to; the greater the amount of constant ventilation, the better. During an epidemic, and particularly when erysipelas, pyæmia, local gangrene or parotid swellings are common complications, the walls ought to be frequently lime-washed. Injecting showers of diluted Condy's fluid, from a large garden syringe, through the ward, I have found to have a marked effect in purifying the atmosphere; but no method of fumigation must be allowed to interfere with the abundant admission of fresh air.

The bedding, used by a typhus patient, ought to be taken to pieces, thoroughly washed, and baked, and then exposed to the air. Where this cannot be done, it ought to be destroyed. The bedstead should be washed with a solution of Condy's fluid, or of chloride of lime. In general hospitals, the same bed and bedding ought always to be reserved for typhus cases.

Before his discharge from the hospital, each patient should have a warm bath, and afterwards put on his purified clothes.

Friends, who visit the sick, should be prevented sitting on their beds, or approaching so close, as to inhale their breath or the emanations from beneath the bed-clothes. All unnecessary visits are to be prohibited.

In a private house, after the patient's recovery, the walls and ceiling of the room ought to be scraped and whitewashed or re-papered, the floor and furniture washed first with a weak solution of Condy's fluid, or of chloride of lime, and afterwards with soap and water, and the doors and windows kept open night and day for a week. At the end of this time the room may be re-inhabited with safety.

Not only must every measure be taken to destroy the typhus-poison, but all those agencies, which are known to predispose the system to its influence, must be avoided. Of these, the most powerful is debility from deficient food or from other causes. The Guardians

of the poor and the Commissariat departments of armies and navies ought to be impressed with the fact, that a nutritious diet is one of the best preventives of typhus. Nurses and other attendants on the sick should have a liberal diet, and ought never to visit the wards with an empty stomach, while the opposite error of freely indulging in ardent spirits, in the mistaken notion of warding off the fever, is equally to be deprecated.

Nurses and attendants on the sick should have ample time for sleep; they ought never to sleep in the sick room, and should be made to take exercise daily in the open air. Fatigue of mind or of body is to be scrupulously shunned by persons, who are necessarily exposed to the poison of typhus. In the case of hospital nurses, occasional recreation is no less necessary for keeping up their spirits, than for encouraging them in their dangerous duties.

Personal cleanliness, frequent bathing, and frequent changes of under-clothing ought to be enjoined on every person who is exposed to typhus.

Abundant evidence might be collected, to demonstrate the efficacy of the measures here recommended, for preventing the propagation of typhus.

B. *Curative Treatment.*

The curative treatment of typhus is both hygienic and therapeutic.

1. *Hygienic Treatment.*

A typhus patient is to be placed in a large, airy room, from which the carpet, hangings, and all unnecessary articles of furniture have been removed. The temperature ought to average 60° Fahr. Cold is less injurious than excessive heat, for cold air contains more oxygen than heated air. Thorough ventilation must be secured by open doors and windows, care being taken not to expose the patient to a direct draught of cold air. In this way, a ready escape is afforded to the noxious emanations from the body, by which the disease is propagated to others, and the inhalation of which, with the atmospheric air, may aggravate the disease in the patients themselves. The relative advantages of isolating cases of typhus, and interspersing them in the wards of a general hospital, is a subject on which some difference of opinion exists, and of such vast importance, both as regards the patients and their attendants, that it will be considered in a separate chapter. Meanwhile, it may be stated, in support of the good effects of ventilation, that, on several occasions, the mortality has been found considerably less among cases treated in temporary

sheds and tents, than among those treated, at the same time, in crowded hospitals. This was the case in Dublin in 1826,[p] and in Edinburgh in 1847.[q] Steele also called attention to the remarkable exemption of the cases treated in sheds at Glasgow, in 1847, from erysipelas, which, at the time, was a common complication in the infirmary wards.[r]

Bright light is to be excluded from the patient's eyes; but his room ought not to be too much darkened during the day. The proper alternation of day and night greatly conduces to sleep. The room is to be kept perfectly quiet, and all necessary communications are to be made, in a clear and distinct voice. Nothing annoys or excites sensitive patients more, than to hear whispering going on in the room. Care ought also to be taken not to contradict the patient in his delirium; to do so, or to attempt to reason with him, only increases his excitability. Every effort should be made to cheer him, and to prevent him from desponding; gloomy looks, on the part of the attendants, may cause him to forebode a fatal event, and such foreboding in itself exercises an unfavourable influence on the result.

Beds.—A hard mattress, or a spring-bed, is the best. Soft feather-beds are objectionable; they require more attention, to keep them comfortable, while they heat the patient, and make it difficult for the attendants to shift him. I have known them extensively used at the London Fever Hospital, and I have no reason to believe that they obviate bed-sores; indeed, the pressure exerted by compressed feathers is probably as great as that from hair or flock. In public practice, feather-beds are also objectionable on the ground of expense. In the event of bed-sores forming, recourse must be had to the water-bed. In every case, it is well to cover the mattress with a large sheet of water-proof cloth. In private practice, the patient will often derive great comfort from having two beds in the room, and from being changed from one to the other, once or twice in the twenty-four hours. The change will sometimes secure sleep, after other measures have failed.

As soon as the disease has declared itself, the patient must be put to bed, and every exertion looked upon as a drain on his store of strength. Patients who struggle against the disease, and exert themselves at the commencement, usually suffer from great prostration afterwards. After the first week of the disease, the patients ought to be provided with a bed-pan, and on no account permitted to get out of bed. When they become delirious, and attempt to

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[p] O'Brien, 1828. [q] R. Paterson, 1848. [r] Steele, 1848.

get out of bed, it is too often the custom with nurses to fasten them down, or to apply the strait-waistcoat. This should never be done, except in extreme cases. The feeling of restraint increases the patient's efforts to get loose, while his fruitless efforts augment the muscular debility, and add to his mental sufferings. The patient must be kept in bed by a strong and vigilant nurse, on whose part, gentleness and kind words will often avail more, than physical force.

Sponging. The free escape of the cutaneous exhalations ought to be encouraged by sponging the body, twice, or three times, daily, with tepid water, which may be advantageously mixed with small quantities of Condy's Fluid or of Muriatic Acid (ʒj. ad. Oj.). The patient often experiences the greatest comfort and benefit from this practice, which also diminishes the risk to the attendants, by preventing the accumulation of poisonous exhalations.

Diet. Of the many evils which sprang from the notion, that the symptoms of typhus were due to cerebral inflammation, one of the greatest was a starving system of treatment. No one contributed to overthrow this system more, than the late Dr. Graves. 'If,' said he, 'you are at a loss for an epitaph, to be placed on my 'tomb, here is one for you: He fed fevers.' There can be little doubt, that many persons have fallen victims to prolonged abstinence during typhus, to say nothing of the starvation which, in the poor, often precedes the attack. So far from delirium, stupor, and the other cerebral symptoms of typhus counter-indicating food, it has been shown that these symptoms may result from starvation.[p] Nourishment must be pressed on the patient, even if he seems to have little or no inclination for it; it must be remembered, that his sensibilities are blunted, and that he is incapable of judging of what is good for him. But, inasmuch as the power of digestion is greatly impaired, much care and judgment are required, in the selection of food and in its mode of administration. Nourishment ought to be given often, at stated intervals; at least once every three or four hours, after the fourth day of the fever. If the patient remains long in a state of drowsy stupor, he ought to be roused at fixed hours to take food and stimulants; but if, after much restlessness, he falls into a quiet sleep, he ought not to be awakened, because the hour for food has come round. There is reason to fear, that the tendency of modern practice in England is not to starve fevers, but to over-feed them. I am persuaded that incalculable injury is sometimes inflicted, by forcing food down the patient's

[p] GRAVES, 1848, i. 119.

throat every half-hour or every quarter of an hour. The patient is not allowed a moment's peace, while the food is not absorbed, but is discharged little changed from the bowels, and often induces diarrhœa and tympanitis. As a rule, it is unnecessary to administer food oftener than once in two hours. The food ought to be both nutritious and digestible, and when the tongue is dry and brown, all solids must be prohibited. In the morning, at nine, a cup of coffee and milk with dry toast, or milk alone, may be given; and at noon, a basin of strong beef-tea or chicken-tea, to which may be added arrowroot, vermicelli, or gelatine. At three o'clock, the beef- or chicken-tea may be repeated, or arrowroot made with water, milk, or port wine, substituted. At six, another cup of coffee,[q] with toast may be given; and at nine o'clock, a tumbler of wine-whey, or of wine-whey beaten up with egg. Beef-tea, arrowroot, and wine-whey are to be given three or four times in the course of the night, while wine, brandy, and drinks are to be administered along with, and in the intervals of, the meals, according to circumstances. As soon as the appetite returns, or if the tongue is moist, calf's-foot jelly, chicken-panada, port-wine jelly, custard-, sago-, tapioca-, or arrowroot-pudding, or stewed apples, may be allowed. It is well to prohibit uncooked fruits of all sorts, during an attack of typhus, as they often produce tormina, flatulence, or troublesome diarrhœa.

Drinks. Patients are very capricious in their choice of drinks, and, in private practice, the medical attendant must be prepared to humour them with a variety. Barley-water, toast-water, water-gruel, orangeade, lemonade, apple-water, tamarind-water, currant-jelly-water, raspberry-vinegar, soda-water, and barley-water treated with a few drops of mineral acid, or with bitartrate of potash (ʒij. ad Oj.) may be tried; but, by the end of the first week, the patient usually loathes them all, except pure water and soda-water. There is no objection to the patient drinking often; but he ought not to be allowed more than a wine-glassful at a time. A small piece of ice in the drink will often allay thirst, and is always greatly relished when the thirst is excessive; the combination of a weak infusion of some bitter substance, such as cascarilla or quassia, will often assuage it, after other measures have failed. According to Lyons, camphor is often a specific against thirst; it may be given in the form of camphor mixture or of Murray's fluid camphor.[r]

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[q] The use of coffee, as a medicinal agent, will be subsequently alluded to.

[r] Lyons, 1861, p. 202.

A Nurse. One of the most essential points in the treatment of typhus is securing the services of a kind, experienced, and judicious nurse, strong enough to lift the patient when necessary. The friends or relatives of the patient ought never to be permitted to supply the place of a regular nurse. 'Affection and sorrow,' says Dr. Graves, 'cloud the judgment, and the mistaken tender-'ness of relatives, their want of due firmness, presence of mind, 'and experience, will frequently counteract your exertions, and 'mar your best efforts.' A thousand duties devolve on a nurse, which a nurse alone can perform. The mere moving or raising the patient in bed, and changing his linen, are duties performed very differently by a nurse and an inexperienced person; and no one appreciates more readily, than even the delirious patient, the tenderness and skill of those who minister to his bodily wants. The nurse ought to note, on a piece of paper (presented to the physician at each visit), the hours at which food or medicine has been administered, or at which any remarkable change in the symptoms has occurred. In hospital practice, these notes may be written on a card suspended at the head of the bed.

2. *Therapeutic Treatment.*

It may be well to allude, in the first place, to several methods of treatment, which have been recommended, and afterwards to mention those principles, which are indicated by modern pathology and experience, or what appears to be the Rational Method of Treatment.

Bloodletting. As typhus is essentially a disease of debility, it may appear surprising that general bloodletting, to a large amount, was long a favourite remedy, with many practitioners in this country. Most modern physicians would regard such a practice as almost fatal; and probably none of its former supporters would venture to have recourse to it at the present day. Modern observation has shown that the effect of bloodletting in typhus, is to increase the mortality; while even in the patients who recover after it, the nervous symptoms occur sooner, and with greater intensity, and are of longer duration, the eruption is darker and more copious, and convalescence is greatly retarded.[8] The great revolution in medical practice, within the last twenty years, both in idiopathic fevers and in acute inflammations, has lately attracted much attention, and it has been the fashion to ascribe it to a change in the type of disease, necessitating a corresponding

[8] See, for example, HALLER, 1853.

change in the principles of treatment. Continued fevers have been one of the fields on which the battle of change of type has been fought; but a careful study of their history fails, in my opinion, to lend any support to the theory in question.

In the first place, it is well to observe, that, prior to the commencement of the present century, the practice of all the best observers did not indicate any change of type in typhus. If we turn to the accounts given by Fracastorius, Hoffmann, Rogers, Strother, O'Connell, Wall, Pringle, Lind, Smyth, Willan, and many others,[t] we find that bloodletting was almost universally condemned. The practice of bleeding originated in the erroneous theories of Clutterbuck and Armstrong, already alluded to; and the success of the practice appeared to be established from the circumstance, that it was proposed shortly before an epidemic, consisting, for the most part, of relapsing fever, the mortality from which, with, or without bloodletting, is much less than that of typhus. After this, practitioners were unwilling to relinquish a remedy, which, in the epidemic of 1817-19, appeared to be attended with signal success, as compared with the previous treatment of true typhus, believed to be the same disease. But, by and bye, as typhus was again substituted for relapsing fever, and more especially as the careful study of morbid anatomy exposed the erroneous doctrines of Clutterbuck and Armstrong, bleeding was again condemned in the treatment of typhus, and practitioners attributed the change in their practice to a change in the type of the disease. The change, however, was not one of type, but of disease. In the next chapter, it will be shown, that even relapsing fever, recognized as distinct from typhus, is best treated without bleeding. In typhus, prostration is one of the chief dangers to be apprehended, and this will certainly be hastened and aggravated by the loss of even a small quantity of blood, while the greatest depletion has never succeeded in arresting the disease. That headache and other distressing symptoms may sometimes be alleviated by bloodletting, there can be no doubt; but the powers of the system must not be lowered for such an object. Even local depletion is never permissible, except in rare cases, afterwards mentioned.

Alcoholic Stimulants. The history of alcoholic stimulants in the treatment of typhus, is the reverse of that of bloodletting. Almost all the writers of last century recommended them; and some were in the habit of prescribing them in large quantities.[u] During the

[t] See *Historical Account.* [u] See page 33.

reign of bloodletting, extending over the first quarter of the present century, stimulants were seldom, and sparingly employed; but for the last thirty years, mainly through the teaching of Alison, Graves, and Stokes, they have again constituted an important part of the treatment of most practitioners.

At the present day, there is reason to fear, that many practitioners are in the habit of administering alcohol in fever, in too large quantities, and in too indiscriminate a manner, a quick pulse or cerebral symptoms being regarded as certain indications for its use, even at an early stage of the disease. It is not uncommon for 18, 24, 36, or even upwards of 48 ounces of brandy to be given, in divided doses, in twenty-four hours. This is done in the belief that the alcohol nourishes the body. According to the late Dr. Todd, alcohol in fever, is to be viewed as an article of food, rather than as a medicinal stimulant; it is transformed in the system and affords pabulum appropriate to the direct nourishment and preservation of nervous tissue, and to the upholding of the calorifacient process, being eliminated in the form of carbonic acid and water, and not as unchanged alcohol.[x]

While it has been shown by statistical data,[y] that the systematic treatment of fever with large quantities of alcohol is not remarkable for its success, the rate of mortality being even above the average, it comes to be an important question, of late much discussed, whether alcohol acts as an article of food, or as a medicinal stimulant. On the one hand, it is contended, that it undergoes chemical transformation in the system, and contributes to nutrition and the maintenance of animal heat, that when large quantities in divided doses are given, it cannot be smelt in the breath, and that, in acute diseases, it is capable of sustaining life, without the help of any other food. Such are the statements made by many excellent observers.[z] On the other hand, it is argued, that alcohol is not transformed in the human body, but that it is eliminated unchanged with the various excretions, and that, consequently, it is not a food, and acts only as a medicine.[a]

Although the statement, that *the whole* of the alcohol taken into the system is eliminated unchanged, appears to me to have been made too confidently, and to be far from being proved, the researches

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[x] TODD, 1860, p. 459. [y] *Brit. & For. Med. Chir. Rev.* Oct. 1860, and *Lancet*, Nov. 1860. [z] See, for example, a paper on 'The Alcohol Question,' in the *London Medical Review*, 1862. [a] Dr. EDWARD SMITH, *Brit. Med. Journ.* Nov. 2 and 16, 1861; *Trans. Med Soc. of Lond.* Jan. 14, 1861; and *Journ. of Soc. of Arts*, Jan. 18, 1861.

of Messrs. Perrin, Duroy, Lallemand,[b] and Edward Smith,[c] certainly show, that alcohol is excreted in considerable quantity by the skin, kidneys, and bowels; and hence, the absence of alcoholic odour from the breath,[d] does not prove that the alcohol has been transformed; it is possibly often passed in large quantity by the bowels, without being absorbed, the power of absorption being impaired in consequence of the disease. But, admitting that alcohol may be partly transformed, and help to support combustion and animal heat, the fact of its containing no nitrogen renders it unfit for nourishing those very tissues, which suffer the greatest waste in fever, or indeed of supporting life. The cases where it has been said to have supported life for many days, without any other food, may be partly due to the power, which it is said to possess, of arresting the destructive metamorphosis of tissue, but must be viewed in connection with the fact, that life may be sustained for weeks on nothing but water.[e] The opinion, that alcohol *directly nourishes and preserves* nerve tissue, is ingenious, but has little foundation.

Again, it must be remembered, that alcohol, in over-doses, is a poison. Although, it may not excite local inflammations, even in large doses, it deranges nutrition, lessens the secretions, diminishes the amount of urinary water, and so impedes the elimination of urea. It is apt to induce a state of coma, which, when added to the coma resulting from the disease, must greatly add to the dangers and difficulties of the case.

After a careful study of all that has been written on the subject, and observations of many hundreds of cases, I cannot admit that the beneficial action of alcohol in fever, powerful though it be, is that of *food*. It seems to me more reasonable to adhere to the old view, that it is a medicine, which, like many other medicines, is poisonous in an over-dose. Given as a medicine, it stimulates the nervous system and the organs of circulation, and it probably checks the destructive metamorphosis of tissue,[h] its immediate effects being, to increase the force of the heart, to promote the capillary and cerebral circulation, and so to remove delirium depending on per-

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[b] *Compt. Rend.* Oct. 1859, and *L'Union Méd.* 1859, No. 127.

[c] *Trans. of Lond. Med. Soc.* 1861; *Journ. of Society of Arts*, 1861.

[d] According to my experience, the absence of alcoholic odour from the breath, when fever patients are taking, even no more than ten ounces of brandy in 24 hours, is far from being so common as has been stated.

[e] CARPENTER'S *Princip. of Hum. Phys.* 5th ed. p. 59.

[h] BÖCKER and HAMMOND'S experiments seem to show, that alcohol has the power of retarding the formation of urea. See PARKES, *On the Urine*, 1860, p. 72; also *Brit. & For. Med. Chir. Rev.* April, 1858, p. 315.

verted cerebral nutrition. Hence the cardiac and radial pulses are the grand criteria for guiding us, in the administration of alcohol in fever, not the frequency of the beats, but their strength. As long as they exhibit no tendency to diminish in strength, alcohol is unnecessary, and may be injurious; but when they flag, alcohol is our best and surest remedy. It is perfectly true, that when stimulants act beneficially in fever, they often reduce the frequency of the pulse, quiet delirium, and are followed by improvement in all the symptoms; but I do not hesitate to say, that there are certain forms of delirium in typhus (e.g. *delirium ferox*), independent of cerebral inflammation, where alcohol has often a precisely opposite effect and does harm, and that flushing of the face, increased frequency of pulse, and increased delirium, after giving alcohol, are contra-indications, rather than indications for increased administration.[i]

More minute instructions for the administration of alcohol and wine in fever will be given hereafter; but it may be here mentioned that the profession is indebted to Dr. Stokes,[k] for pointing out the importance of the cardiac and radial pulses, as guides for the use of alcohol in fever.

The Cold Affusion. Towards the end of last century (1787), the cold affusion was proposed by Dr. Currie[l] of Liverpool, both for arresting and mitigating continued fever. The patient was seated naked, in an empty tub or bath, and several buckets of water, of a temperature of 40° to 50° Fahr., were poured from a height of from one to three feet, or more, over the head and chest. He was then hastily dried and restored to bed; and in most cases the operation was repeated once or twice daily. It was stated, that, in many cases, if resorted to during the first three days, this treatment arrested the disease, while in others it reduced the pulse and temperature, relieved many of the distressing symptoms, and particularly the headache, restlessness, and delirium, and conducted the disease to a safer and speedier issue. The affusions were employed, at any stage of the fever; but the effects were always most salutary at an early stage. They were said to be contra-indicated, however, when the temperature of the skin, ascertained by the thermometer, was not above the normal standard, or when, notwithstanding an elevation of temperature, the patient complained of chilliness, or suffered from severe diarrhœa or profuse

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[i] See a valuable paper by Dr. W. T. GAIRDNER, on the *Use of Alcoholic Stimulants, in Hospital Practice, Edin. Med. Journ.* May, 1861.

[k] STOKES, 1839. [l] CURRIE, 1797.

sweating. The wonderful results obtained by Currie, were confirmed by numerous observers, in different parts of the world, whose testimony is recorded in the third edition of his work, published in 1805. But in the British epidemic of 1817-19, the practice was followed by many with great perseverance, and the general results, according to Dr. Christison, were, that in very few, if any, was the disease arrested by it; that although an abatement of febrile heat and restlessness occurred almost invariably, it was of short duration, and not to be made permanent by any frequency of repetition; that as much good eventually was attained, by frequent cold or tepid sponging, together with cold applied to the head; and that often the cold affusion occasioned, for a short time after each application, an intense feeling of pressure and weighty pain in the centre of the brain, which could not be regarded without some uneasiness.[m] The results alluded to by the learned author of the article, 'Fever,' in the 'Library of Medicine,' and perhaps the natural aversion of professional men to anything approaching a system of quackery, which makes the water-cure a *panacea* for all diseases, aided by popular prejudice, account for the present neglect of the cold affusion in fevers. But, if there be any truth in hydropathy, available for the relief of human suffering, there ought to be no hesitation, on the part of the regular physician, in adopting it.[n] Many observers, since the time of Dr. Currie, have found the cold affusion a powerful remedy for good, in fevers. In 1820, Dr. Ross, of Leith, found it of great service in 'producing a remission and abatement of the violence of the fever.'[o] In 1830, Dr. Southwood Smith recommended the cold douche, as a most certain remedy for allaying the headache of typhus. The manner in which he applied it, was as follows:— The patient was seated in a large tub; a table was placed at the side of the tub, upon which a man stood, and from as great an elevation as his arms could reach, poured upon the naked head of the patient a steady but continued stream of cold or iced water, from a watering pot without the rose, the stream being made to fall as nearly as possible upon the same spot.[p] Abroad, Horn, Récamier, Blache, and many others, have also obtained excellent results from the cold affusion in fever.[q]

A few years ago, the hydropathic treatment of continued fevers was investigated by Dr. Armitage, who published some very careful observations of cases, in proof of its beneficial effects, and

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[m] Christison, 1840. [n] See *Brit. & For. Med. Rev.* Oct. 1846, p. 446.
[o] Ross, 1820. [p] Smith, 1830, p. 400. [q] Barrallier, 1861, p. 164.

who endeavoured to show, that the cold douche operates not only as a *cooling agent*, but more especially as a *stimulant*, and that it is especially useful in cases where, along with considerable elevation of temperature, there is great stupor, and but little irritability of the nervous system. He found it to be a most effectual measure, for rousing the patient from stupor.[r]

There is no evidence, that the cold affusion ever cut short a case of true typhus; but it is probably a powerful remedy for mitigating symptoms, and too much neglected in modern practice.

Quinine in large doses. Cinchona was introduced into the treatment of typhus by Dr. Miller of London, in 1770,[s] and was afterwards recommended by John Clark,[t] Hildenbrand,[u] and many others. Bateman, however, denounced it as positively hurtful.[x] Gerhard thought that repeated doses of quinine, to the amount of twelve grains in twenty-four hours, were attended by obvious advantages, in the Philadelphia epidemic of 1836.[y]

In 1851, Dr. Robert Dundas[z] announced, that typhus, like intermittent fever, might be cut short by the administration of large doses of quinine. The plan recommended was as follows:— After an emetic, ten grains of quinine were given every two hours, until the symptoms subsided, or until deafness and ringing in the ears supervened, when the remedy was to be discontinued, to be recommenced after an interval of twenty-four hours. After this announcement, Dr. Goolden[a] of London, Dr. M'Evers of Cork,[b] Messrs. Hayward,[c] Gee and Eddowes,[d] of Liverpool, and Mr. Fletcher,[e] of Manchester, reported the results of their use of quinine in numerous cases of fever (typhus and typhoid), as highly satisfactory. But about the same time, or soon after, Dr. Dundas's treatment was tried by Drs. Bennett,[f] Christison,[g] and W. Robertson,[g] of Edinburgh, by Drs. Peacock[h] and Barclay,[i] of London, by Dr. Corrigan,[k] of Dublin, and by Haller,[l] of Vienna, who came to the conclusion that large doses of quinine, so far from ever arresting the fever, not unfrequently gave rise to alarming symptoms. As clinical clerk, I had an opportunity of watching Dr. Bennett's cases, and can confirm the unfavourable opinion which he expressed. The remedy has also been tried in a considerable number

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[r] Armitage, 1852.

[s] *Observations on the prevailing Diseases of Great Britain*, 1770.

[t] Clark, 1802. [u] Hildenbrand, 1811. [x] Bateman, 1819. [y] Gerhard, 1837.

[z] Dundas, 1851 and 1852. [a] Ibid, 1852, p. 417. [b] M'Evers, 1852.

[c] Hayward, 1852. [d] Gee and Eddowes, 1853. [e] Fletcher, 1853.

[f] Bennett, 1852. [g] Ibid. *Clinical Lectures*, 2nd ed. 1858, p. 881.

[h] Peacock, 1856, No. 2. [i] Barclay, 1853. [k] Corrigan, 1853, p. 78.

[l] Haller, 1853.

of cases at the London Fever Hospital, but with no better success. Huss likewise failed to obtain any advantage from it at Stockholm.[m] The bad consequences, observed in some cases to follow its use, have consisted in a marked increase of the coma and delirium, and great depression of the vital powers; and in more than one case it seemed to hasten the fatal termination.

Dr. Dundas's recommendation was accounted for by his belief, that typhus and intermittent fevers were the same disease. This belief was founded on his observation of fever in Brazil, where he maintained that typhus was met with, along with remittent and intermittent fevers, in the same family, at one time, and that these fevers merged insensibly into one another. That this is the case with intermittent and remittent fevers there is no doubt; but it is probable that Dr. Dundas committed the common error of mistaking adynamic remittent fever for typhus. As already stated, there is as yet, no reliable evidence of the existence of true typhus, within the tropics, and none is to be found in Dr. Dundas's work. (See page 58).

With regard to the favourable results said to have been obtained from large doses of quinine in the continued fevers of this country, it may be observed, that few of the cases are given in sufficient detail to enable us to form an opinion as to the nature of its effects, or as to the severity of the case. One thing is certain, that there is no proof that quinine can *arrest* the course of true typhus.

The uselessness of large doses of quinine in the treatment of typhus was confirmed by the experience of the Crimean war. At first, it is stated, that there was a perfect rage for its employment among the French medical officers who had served in Algeria, and who, no doubt, like Dr. Dundas, saw in typhus a strong resemblance to the adynamic remittent fever, to which they had been accustomed. Their expectations were disappointed. Jacquot states that the treatment was tried on the largest scale, and was found not only perfectly useless, but positively dangerous.[n]

Since the above was written, the plan of treating typhus, in its early stage, with an emetic, followed by large doses of quinine, has been advocated by Barrallier of Toulon,[o] who found that it relieved headache, and effected an improvement in the general symptoms. Barrallier, however, says nothing as to the disease being arrested

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[m] Huss, 1855, p. 180. [n] Jacquot, 1858, p. 260.
[o] Barrallier, 1861, pp. 153, 288.

by such treatment, and it may be doubted, if the cessation of headache was not due to the natural course of the disease. (See p. 150).

Warburg's Tincture. Typhus is one of the many diseases for which the celebrated Warburg's Tincture has been recommended. This medicine is said to contain aloes, camphor, and saffron, together with a bitter alkaloid, either bebeerine, or quinine. I have tried it in several cases, according to the instructions laid down by Mr. Warburg, to whom I am indebted for the preparation employed. Half-an-ounce was given at first, and repeated in three hours, and afterwards one drachm was given every three hours. Profuse perspiration usually followed the second large dose; but in none of the cases, did it reduce the pulse, or diminish the temperature (as ascertained by the thermometer), or in any way modify, or abbreviate, the course of the disease. Some cases, apparently of a mild character, where it was given at an early stage, afterwards assumed a severe form. Whatever may be the virtues of Warburg's drops in malarious fevers, in true typhus I believe it to be useless.

Emetics have been recommended, in the early stage of typhus, by most writers, and, in later times, particularly by Hildenbrand, Graves, and Barrallier, with the object of cutting short the fever, or of rendering its course milder. It is very doubtful, however, if true typhus has ever been cut short by an emetic. Graves allows, that the remedy is only of service for this object, if given within the first twenty-four or thirty-six hours of the disease,[p] and at this early stage, before the appearance of the eruption, it is impossible to predict, that a febrile attack will run the ordinary course of typhus. It is not uncommon for persons exposed to the poison of typhus, to be seized with febrile symptoms of some severity, terminating spontaneously in three or four days; if an emetic had been given in such a case, the cure would be attributed to it. At the same time, an emetic is often of undoubted service in relieving symptoms during the first five or six days of the disease. Its good effects are often most marked, in mitigating or removing the headache and general pains, in reducing the temperature, quenching the thirst, and quieting any gastric disturbance. It is only contra-indicated when the patient is unusually weak, or when the disease has advanced beyond the first week.

Purgatives. The systematic employment of purgatives in the treatment of typhus, was first introduced by Dr. James Hamilton of Edinburgh, at the commencement of the present century,[q] and,

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[p] GRAVES, 1848, i. 138. [q] HAMILTON, 1805, pp. 14, 159.

for many years, was an almost universal practice among British physicians. It was thought, that by the free evacuation of the offensive contents of the bowels, the febrile reaction was reduced, and the other symptoms relieved. The bad effects of excessive purging were exposed by Graves, Corrigan, and others, and the practice is now obsolete. Although regular action of the bowels is indispensable, I have repeatedly known alarming prostration caused by diarrhœa, following the incautious administration of purgatives.

Diaphoretics were formerly much employed in the treatment of typhus, and some practitioners still place much faith in them. The most common preparations have been large doses of the liquor ammoniæ acetatis, with or without the addition of small doses of tartar emetic ($\frac{1}{8}$ to $\frac{1}{12}$ gr.), or three or four grains of James's Powder, with a like quantity of Dover's Powder, and of Hydrargyrum c. Creta. After an extensive use of these remedies, I cannot say that I have obtained very satisfactory results from them. Even when diaphoresis is produced, there is no necessary improvement in the general symptoms, while profuse sweating is often followed by increased prostration. At the same time, I have observed great benefit from frequent sponging of the surface, and in cases of profound stupor, with, or without albuminous urine, the propriety of diaphoretic measures may be fairly entertained.

Salines. It was at one time the practice to administer salines in fever, on the erroneous supposition, that febrile symptoms depended on a loss of the saline ingredients of the blood.[r] They are never now prescribed except as *placebos.*

Antimony. Although under certain circumstances, hereafter specified, antimony is a remedy of undoubted utility in typhus, it must always be given with great caution, for fear of inducing a dangerous degree of debility. No practitioner would now think of prescribing it in the way recommended by Rasori, who gave it in every case, and often to the extent of six or eight grains in twenty-four hours.[s]

Mercury is another remedy, once much employed in the treatment of typhus.[t] I have seen many cases treated with it, but never with the slightest benefit. It has been shown by Graves, that pytalism not only fails to relieve the symptoms, or to shorten the progress of typhus, but that it does not protect the system from being attacked.

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[r] See Christison, 1840, p. 183; Tweedie, 1860, p. 589. [s] Rasori, 1813, pp. 25, 37.
[t] Smith, 1830, p. 396; Roupell, 1839, p. 245.

Rational Method of Treatment.

In the treatment of typhus, medicines can do much to relieve symptoms, and to conduct the case to a favourable termination; but, as far as we yet know, they are powerless in arresting its progress, or in shortening its duration. Although many practitioners have, at different times, proposed to cut short an attack of typhus by such heroic remedies as blood-letting, the cold affusion, emetics, and quinine, we possess as yet no such specific. In an admirable essay, published in 1802, Dr. W. Brown of Edinburgh showed, that the power of medicine in arresting or shortening typhus was extremely doubtful.[u] Hildenbrand, in his day, observed: 'No method yet 'known, whether rational or empirical, can cure the contagious 'typhus, either in a direct or in an indirect manner, nor even 'abridge its ordinary and natural course, which is about fourteen 'days.'[x] In our own times, Dr. Stokes speaks equally strongly: 'The treatment of fever,' he says, 'is reduced to a formula. 'We cannot cure fever. No man ever cured fever. It will 'cure itself. If you keep the patient till the fourteenth, the 'eighteenth, or the twenty-first day, he will recover.'[y] Our objects, in the treatment of typhus, should be:—1. To neutralize the poison, and to correct the morbid state of the blood. 2. To eliminate the poison and the products of the destructive metamorphosis of tissue. 3. To reduce the temperature. 4. To sustain the vital powers, and to obviate the tendency to death. 5. To relieve the distressing symptoms. And, 6. To avert and attack local complications. By such means, nature is assisted in effecting a cure; for it is wrong to imagine that it is a matter of indifference, what plan of treatment is adopted.

1. If the opinion be correct, that the altered condition of the blood in typhus is due to the presence of ammonia, either derived from the original poison, or from the products of the destructive metamorphosis of tissue (*e. g.* urea), the first of the above indications will be most readily fulfilled, by the administration of mineral acids. But, whether acids act as antidotes, as alteratives of the blood, or as tonics, their beneficial effects in typhus are, in my opinion, undoubted; and it is curious to observe, that they have been recommended for this disease in all countries, since the days of Forestus, Sydenham, Van Swieten, and Boerhaave. They have lately been highly praised by Huss, of Stockholm;[z] by Haller, of

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[u] Brown, 1802. [x] Hildenbrand, 1811, p. 149. [y] Stokes, 1854.
1818 [z] Huss, 1855, pp. 141, 168.

Vienna ;[a] and by Drs. F. W. Mackenzie,[b] Chambers,[c] and Richardson[d] in our own country. Huss gives preference to the phosphoric acid, in doses of 10 to 15 drops every second hour,[e] on the ground that it is not only a tonic and stimulant in common with other acids, but that the phosphorus exerts a special influence on the central organs of the nervous system. In the more advanced stage of the malady, and particularly if numerous petechiæ and ecchymoses, or profuse sweating, be present, he recommends the substitution of sulphuric acid, in doses of 15 to 20 drops every hour or every second hour.[f] Sulphuric acid, also, it may be observed, is the chief constituent of the *Elixir Acidi Halleri*,[g] so commonly employed in Germany in the treatment of typhus and allied diseases. Drs. Mackenzie and Chambers prefer hydrochloric acid; they direct one fluid ounce of dilute hydrochloric acid to be taken daily in a quart of barley- or plain water, sweetened with syrup of ginger, and flavoured with lemon-peel. Dr. Richardson also gives preference to hydrochloric acid, owing to its less fixed character. My friend and colleague, Dr. A. P. Stewart, tells me that he has employed the *Tinctura Muriatis Ferri* with great advantage, in doses of half-a-drachm every three hours. I have tried the mineral acids in many hundreds of cases during the last few years, and I believe them superior to any other method of treatment, although I am far from ascribing to them the wonderful virtues which some writers have done. I usually commence by ordering the hydrochloric and nitric acids, 20 minims of the former and 10 of the latter, every three hours, each dose being diluted with the patient's drink. A small quantity of simple syrup, or of tincture of orange-peel, may be added, according to the taste of the patient. In the advanced stage of severe cases, when the 'typhoid state' is developed in a marked form, I prefer the dilute sulphuric acid, in doses of 15 to 20 minims every three hours, in combination with ether and small doses of quinine, as afterwards recommended. I have often observed marked improvement follow the commencement of the acid treatment, at whatever stage of the fever it was tried, and although no wine or brandy was given with it.

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[a] HALLER, 1853. [b] *Path. & Treatment of Phlegmasia dolens*, 1862, p. 123.
[c] CHAMBERS, 1858, p. 109. [d] RICHARDSON, 1858, p. 446.
[e] The solution of the Swedish Pharmacopœia contains 25 per cent. of glacial phosphoric acid.
[f] The solution of the Swedish Pharmacopœia contains 11·11 per cent. of pure acid, and has a specific gravity of 1092.
[g] The Elixir Acidi Halleri consists of one-part of concentrated sulphuric acid, to three of rectified spirits. It is given in doses of five to twenty drops, in solution.

Creasote, carbolic acid, the chlorates of potash and of soda, the permanganate of potash (Condy's fluid), and the peroxide of hydrogen are remedies which have been recommended, as antiseptics or as correctives of the blood, in typhus; but after repeated trials of all of them (excepting carbolic acid), I believe them to be very inferior to the acids. With the object of improving the condition of the blood in the advanced stages, I have lately employed, in several cases, the inhalation of oxygen gas from Mr. Barth's apparatus; but I cannot say, that as yet I have observed any marked benefit or change, follow its use.

2. Elimination is to be encouraged by maintaining the action of the kidneys, the bowels, and the skin. From what has been already stated (see pages 13, 146, 162, 174), the importance of maintaining the action of the kidneys, so as to secure the elimination of the products of the exaggerated metamorphosis of tissue, must be obvious. With this object, I am in the habit of allowing the patient to drink water freely, though not much at a time, and of ordering 5 grains of the nitrate of potash with each dose of the nitro-muriatic acid. This addition has also the advantage of keeping up a moderate action of the bowels. Nitre-whey, prepared by boiling ʒij. of nitre in a pint of milk and straining, or the *potus imperialis*, prepared by dissolving ʒj. to ʒij. of bitartrate of potash in a pint of boiling-water, and flavouring with lemon-peel and sugar, may also be used for the same purpose. In the advanced stage, if the patient be very prostrate, or if the bowels be relaxed, nitric ether may be substituted for the nitrate of potash.

Tea and coffee are remedies, which probably deserve to be included under this head. Both have long been recommended as excellent expergefacients in the stupor of typhus;[h] and an observation of Dr. Parkes's renders it very probable that this property is due to their power of eliminating the urea already formed in the blood. Dr. Parkes found that, after administering 120 grains of extract of coffee to a patient in the tenth day of typhus, the total amount of urea excreted by the kidneys in twenty-four hours, which for two days before, and for eight days after, varied from 507 to 552 grains, rose to 723 grains. At the same time,

[h] In 1817, Dr. E. Percival stated, that he had found an infusion of green tea of great service in comatose affections, and especially in those of typhus (*Trans. K. & Q. Coll. of Phys.* 1818, ii.44). His observations were confirmed by Dr. Stoker (1826, p. 110) and by Dr. Graves (1848, i. 123). Strong coffee has long been used on the continent for the same purpose. In 1834, a French physician, clinical assistant to M. Petit, published a number of observations showing the excellent effects of coffee in the stupor and other cerebral symptoms of 'Typhoid Fever.' (*Bib.*1834).

the patient expressed himself as much better, his headache ceased, and his pulse became fuller and stronger.[i] This result is the more remarkable, inasmuch as tea and coffee are known to delay, rather than hasten, the destructive metamorphosis of tissue. Thus the researches of Böcker, L. Lehmann, and Hammond, all agree in showing, that tea and coffee, administered in health, diminish the amount of urea in the urine.[k]

Common salt, or chloride of sodium, was once strongly recommended in the treatment of typhus,[l] more especially when the disease presented putrid or typhoid symptoms, such as great prostration, dry brown tongue, numerous petechiæ, stupor, etc. Its reputed good effects are not to be explained, by its supplying the deficiency of this substance in the blood (see pages 147, 238), but they are possibly due to its antiseptic properties, and to its property of increasing elimination. Bischoff, Boussingault, Knapp, and others, have shown that the effects of chloride of sodium in health is to increase slightly the quantity of urea.[m] It is also to be borne in mind, that the quantity of salt taken with the food is much diminished in fever. Wundt's observations show, that the total removal of salt from the food reduces greatly the quantity of urinary water, and after a few days renders the urine albuminous.[n] For these reasons, I have been in the habit of ordering large quantities of salt to be mixed with the patient's beef-tea, and have found it, in most cases, greatly relished, and occasionally of apparent benefit.

Every remedial agent, which shall be found hereafter to promote the elimination of urea, without increasing the destructive metamorphosis of tissue, will deserve a trial in typhus.

The action of the bowels is to be kept up by emetics and laxatives. If the patient is seen within the first five or six days of the attack, it is well to commence by prescribing an emetic of

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[i] PARKES, 1857; also PARKES *On the Urine*, 1860, p. 259.

[k] See ibid. p. 76.

[l] Chloride of sodium was first recommended in the treatment of fever by Dr. Robert Reid of Dublin, in 1827. In 1832, Dr. Graves began a series of experiments with it, and in 1835, reported to the British Association that he had tried it in many hundreds of cases, and that when there was great prostration with numerous petechiæ and other symptoms of putridity, no remedy acted so energetically. He prescribed 15 to 20 drops of a saturated solution every four hours (GRAVES, 1835). Two years later, Dr. Hudson of Navan, reported that he had given it in 47 cases, 'in every instance with the best effect.' (HUDSON, 1837, p. 351). Salt was also at one time highly praised by Chomel (1834), Dr. Dor of Marseilles (*Gaz. Méd de Paris*, Fév. 28, 1835), and by other French practitioners, in the treatment of enteric fever; but on the whole, it appears to have been of less service than in typhus (See also BARTLETT, 1856, p. 161).

[m] PARKES, 1857; also PARKES *On the Urine*, 1860, p. 65. [n] Ib. p. 85.

ipecachuan (℈j) and antimony (gr.j.), or of carbonate of ammonia (℈ij.), followed, if the bowels remain confined, by a mild laxative of rhubarb and calomel, or of castor oil, or by a simple enema. As a rule, the action of the emetic is followed by relaxation of the bowels. The advantages of emetics, at this stage, have already been pointed out (p. 263). During the progress of the disease, laxatives or enemata are to be repeated, if necessary, so as to secure a motion once daily. As already stated, however, active purging is to be avoided, and, in most cases, a small dose of castor oil, or a simple enema is all that is required.

Although diaphoresis has not been found of much service as a principle of treatment, the action of the skin ought to be kept up by repeated sponging, in the manner already recommended (p. 253).

3. The practice of sponging is a hygienic measure of no small importance: it often affords great relief, and it has the additional advantage of fulfilling the third indication for treatment, that of reducing the temperature. For this object, also, the cold affusion, as practised by Currie and others, deserves a greater share of attention than it has yet received (see page 259).

4. The vital powers are to be sustained by appropriate food (see page 253), and by the administration of alcoholic and other stimulants and tonics. There is reason to believe, that wine and alcohol, in small quantities, as well as tea and coffee, not only have a directly stimulant action on the nervous system and on the organs of circulation, but that they check the destructive metamorphosis of the tissues, and thus stay the progress of interstitial death. It is impossible to lay down any rules for the administration of wine and alcohol in typhus, which will be universally applicable. Their indiscriminate employment as food in fever is a convenient, but a dangerous, practice; few remedies require more discrimination in their use. The following observations are recommended for the guidance of the practitioner; but experience and tact can alone teach the proper times and circumstances, which call for them.

a. There are some cases of typhus which, under a supporting diet and the mineral acids, do well without wine or brandy, at any stage of their course.

b. In few cases is wine required, or of any service, during the first five or six days of the illness; but most cases require a greater or less amount at some time during the second week; and, as a rule, it is good practice, to commence the exhibition of stimulants about the seventh or eighth day.

c. The chief indications for the administration of alcohol are derived from the organs of circulation.

d. Extreme softness and compressibility of the pulse, and more especially an irregular intermitting or imperceptible pulse, are far greater indications for the use of stimulants than mere rapidity. An abnormally slow pulse (*e. g.* 40 to 60) is occasionally a stronger indication for stimulants, than a quick pulse. Sometimes the pulse is thrilling and has a certain degree of hardness, but at the same time it is very compressible, and then stimulants may be expected to do good. If the pulse becomes quicker, and the face flushes, under the use of stimulants, they are contra-indicated; if the pulse is made slower, they may be expected to do good.

e. The state of the heart affords information of great value. Stimulants are not required when the cardiac impulse is good; but when the impulse is weak, and when the first sound is impaired or absent, a liberal allowance is demanded. A diminished, or absent, impulse and first sound are compatible with a distinct radial pulse; and hence, in every case where there are doubts as to the propriety of giving stimulants, we must not trust entirely to the pulse, but examine the heart with the hand and stethoscope (see p. 136).

f. Stimulants are always demanded, when a tendency to syncope, or to great diminution in the strength and volume of the pulse, is produced, by raising the patient to the semi-erect posture.

g. Cæteris paribus, stimulants are more necessary, the darker and more copious the eruption. Numerous petechiæ are an indication for stimulants.

h. A burning, dry skin is in itself an indication against alcohol; whereas profuse perspiration, with no contemporaneous improvement in the general symptoms, calls for an increased supply.

i. Coldness of the extremities is an indication for alcohol. The increased heat that follows its use is not necessarily due to the chemical transformation or combustion of the spirit, but to the increased circulation and diminished evaporation from the skin.

k. The more the case presents the characters of the 'typhoid state' (*i. e.* stupor, low delirium, tremor, subsultus, and involuntary evacuations), the more will stimulants be demanded.

l. Scanty urine, of low specific gravity, containing little urea or much albumen, and complete suppression of urine, are in themselves indications against a large amount of spirits.

m. Delirium must not be regarded as, of necessity, indicating alcoholic stimulants. The propriety of giving stimulants in delirium depends on the state of the pulse. If the patient becomes more restless and delirious under the use of stimulants, they do harm; if he becomes more tranquil, they do good.

n. Alcohol, as a rule, is contra-indicated, if there be severe darting or throbbing headache, or acute noisy delirium, especially when these symptoms co-exist with great heat and drynesss of skin, and suffusion of the eyes, and with little or no impairment of the cardiac and radial pulse. When alcohol is thought to be required, under such circumstances, it should be given in the intervals between the paroxysms of delirium.

o. A dry, brown tongue is an indication for wine and brandy, rather than otherwise; if the tongue become clean and moist at the edges, it is a sign that the alcohol is doing good.

p. The presence of complications, as a rule, increases the necessity for stimulants, and certain complications, as pyæmia, erysipelas, bronchitis, pulmonary hypostasis, pneumonia, inflammatory swellings, bed-sores, and local gangrene, usually demand large quantities.

q. Stimulants are required earlier, and in larger quantity, by persons who have led intemperate lives, and of advanced years.

Port, sherry, madeira, brandy, gin, and whiskey, are the forms in which alcohol is best given; but when a weaker stimulus is wanted, claret and burgundy answer well. Malt liquors are best adapted for convalescence. Spirits contain from 50 to 60 per cent. of alcohol; sherry and port, from 17 to 24 per cent.; and good porter and ales from 6 to 8 per cent. Hence two fluid-ounces of spirits are equal to 5 or 6 of wine. Although some practitioners prefer wine to spirits, it is not certain that the former possess any advantages, apart from the alcohol which they contain.

Spirits ought to be given diluted. An excellent way of giving them is in iced soda-water; but where there is great prostration, and especially where the skin is cold and covered with perspiration, the best stimulant is hot brandy- or whiskey-punch.

As the stimulant effects of wine or spirits are transient, they ought to be given in divided doses frequently repeated. In urgent cases, the dose may be repeated every hour, and, as a rule, a larger quantity will be required during the night, and towards morning, than in the day time, for it is usually in the early morning, that the vital powers are at the lowest ebb. Many patients are undoubtedly lost, from negligence of their attendants at this time.

It is impossible to give any positive instructions, as to the quantity of wine or spirits, required in each case. Usually, it is well to commence with not more than four ounces of wine in twenty-four hours, and to watch its effects. It will rarely be necessary to give more than eight ounces of brandy at any period of the fever. Occasionally, however, this allowance may be

exceeded, and it is astonishing to find what large quantities some patients take with advantage, who have previously led temperate lives. Stokes records the case of a man, who, in twenty-one days, consumed twenty-four bottles of wine, and six bottles of brandy, without exhibiting any signs of intoxication.[o] In my own practice, I never see reason to exceed sixteen ounces of brandy in twenty-four hours, and I believe that the cases where this amount is required are exceptional.

In urgent cases, the food and alcoholic stimulants must be persisted with, as long as the patient is able to swallow; and even when he can no longer swallow, the case is not to be given up. I have known cases where life appeared to be saved, by frequent enemata of beef-tea and brandy, after the patient had ceased to take anything by the mouth.

As soon as the symptoms, for which wine and spirits are given, begin to recede, the quantity ought to be reduced, and smaller doses ordered at longer intervals.

In most cases of great prostration, it is well to combine other stimulants with the wine or spirits. The chief remedies recommended for this purpose are, carbonate of ammonia, the chloric, sulphuric, and nitric, ethers, camphor, and musk. Of these, the carbonate of ammonia is the most commonly employed; and some physicians are in the habit of prescribing, it through the whole course of the fever. Although ammonia is unquestionably a powerful stimulant, I must confess that my experience of it in typhus has not been favourable; and if a super-ammoniacal condition has anything to do with the production of the symptoms, the propriety of giving ammonia as a medicine is doubtful. Moreover, I can confirm the statements made by Drs. Kennedy,[p] Joseph Bell,[q] and Lyons,[r] that when given in repeated doses, it is apt to irritate the bowels, and produce diarrhœa. For these reasons, I prefer the different ethers. Ten to thirty minims of chloric or sulphuric ether, or thirty minims to one drachm of nitric ether, may be added to each dose of the acid mixture.

With these remedies, I am in the habit of combining small doses of Quinine, or of the Liquor Cinchonæ, in some such prescription as the following:

℞—Acid. Hydrochlor. dil. . . . ℥xx.
Acid. Nit. dil. . . . . ℥x.
Sp. Æth. Nit. . . . . ʒj.
Liquor. Cinchonæ . . . . ℥xxx.
Decoct. Scopar. Co. . . . . ℥j
Ft. haust. 3â q.q. horâ sum.

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[o] Stokes, 1854. [p] H. Kennedy, 1860, p. 227. [q] J. Bell, 1860. [r] Lyons, 1861, p. 211.

Or, R. Quinæ Disulph. . . . gr. ½.
Acid. Sulph. dil. . . . ℥xx ad ℥xxx.
Æth. Sulph. . . . ℥xv ad ℥xxx.
Syrup. Aurant. . . . ℥j.
Decoct. Scopar. Co. . . ℥j.
Ft. haust. 3â q.q. horâ sum.

Musk and camphor are also powerful stimulants, and, under certain circumstances, to be presently alluded to, are of great service.

5. The first distressing symptom, which the physician is called on to relieve, is usually *headache*. After the administration of an emetic, and securing the action of the bowels, measures which often afford great relief, all that is necessary in most cases, is to apply cold to the forehead, by means of a single layer of wet lint moistened, as fast as it dries, in water or in some evaporating lotion. But when the headache is very intense, such treatment is of little or no avail. The headache is then often associated with flushing of the face, redness of the conjunctivæ, and dry hot skin, and, if neglected, is apt to be followed by *delirium ferox*. Under such circumstances, the head ought to be shaved, and a bullock's bladder filled with ice tied over the scalp, or recourse must be had to the cold affusion. This may be administered by simply bringing the patient's head over a basin at the edge of the bed, and pouring cold water (40° to 50° Fahr.) on it, from a height of two or three feet; but to do it effectually, we must follow the instructions given at page 260. The relief obtained by this treatment is often immediate and complete. If the headache returns, the affusion ought to be repeated.

In cases of intense headache, where the patient is young and robust, I have been repeatedly tempted to apply from two to four leeches to the temples, and I have known their application followed by complete and permanent relief. I have seldom seen much benefit to the headache from blisters or sinapisms to the forehead or nape.

In aged and infirm patients of feeble circulation, caution must be exercised in applying cold to the head, which has often too depressing an effect, and it will be advisable to try the effect of warm fomentation. A double fold of lint, moistened in warm water and vinegar, is to be laid over the scalp and covered with oiled silk, the application to be renewed every three or four hours. Dr. Graves, in his Clinical Lectures, strongly recommends warm fomentations as the best and most efficacious application for the relief of the ordinary headache of fever.[s]

[s] Graves, 1848, i. 163.

Two remedies have been recommended by Barrallier of Toulon, for the headache of typhus: quinine in large doses, and the muriate of ammonia.[t] After an emetic, he orders $2\frac{1}{2}$ or 5 grains of quinine to be given every quarter or half an hour, until 15 or 30 grains have been taken, and if this fails, he gives 46 grains of muriate of ammonia, in three or four doses, at intervals of half an hour, dissolved in water with a little syrup of orange. I have tried Barrallier's treatment in several cases, without ever observing the slightest benefit. In estimating the effects of remedies on the headache of typhus, its natural abatement or cessation about the eighth day must be borne in mind.

Sleeplessness, Nervous Excitement, and Delirium, are among the most important symptoms that require treatment.

Sleeplessness is often complained of from an early stage of the disease, and, if not relieved, greatly exhausts the patient, and is apt to be followed by much delirium. The practitioner cannot be too forcibly impressed with the fact, that loss of sleep, at any stage of typhus, if it continue for two or three nights, is of itself sufficient to kill; and that even the shortest sleep is an advantage to the patient. At the same time, it must be borne in mind, that sleeplessness, as well as the other cerebral symptoms of typhus, are independent of inflammation of the brain, or its membranes, and is not to be remedied by antiphlogistic treatment. The proper treatment for sleeplessness varies with the stage of the disease, and with the nature of the other symptoms. In every case, the practitioner should satisfy himself that the symptom is real, and not imaginary. (See page 156).

When sleeplessness occurs during the first week of the disease, it is usually accompanied by headache; and the measures recommended for the relief of the latter symptom often suffice to procure sleep. If they fail, and the patient has slept little or none for thirty-six hours, recourse should be had to opiates. Ten minims of Battley's Solution, or 15 minims of the solutions of the muriate or acetate of morphia, or 5 grains of the compound soap pill, may be given about nine, p.m.; followed in two hours by half the dose, if the patient does not sleep. If there be great headache, a dry, hot skin, and a pulse of good strength, the opiate will be advantageously combined with antimony, in the manner stated below. I am inclined to think that the employment of opium in typhus is more dreaded than it ought to be. With the precautions to be mentioned, I have never seen any injury, and often the greatest benefit, result from it.

[t] BARRALLIER, 1861, pp. 153, 288.

When delirium and nervous agitation are associated with sleeplessness, much good will be effected by attending to the rules already given for the proper management of the sick-room, and more especially by good ventilation, a proper alternation of light and darkness, and the avoidance of whispering on the part of the attendants. If the hearing be very sensitive, the patient's ears may be stuffed with cotton, as suggested by Dr. Corrigan.[u] At the same time, care must be taken not to disturb him every quarter or half an hour, for the purpose of giving him food and drinks, which he does not require. 'On this point,' Dr. Corrigan justly observes, 'it often appears as if common sense 'had left both the professional and other attendants, who, in 'this way, force upon the nutritive function a quantity of fluid, 'which, even in its healthy state, would be too much for it, and 'which, in its impaired condition, it is incapable of assimilating or 'absorbing.'[x] In the slighter forms of delirium, no further interference is necessary; but when great delirium co-exists with sleeplessness, recourse must be had to other measures, the nature of which must vary, according to the state of the circulation; and as the patient's condition approaches more to that of *delirium ferox* on the one hand, or of *typhomania* or *delirium tremens* on the other. (See page 152).

In the former case, the cardiac and radial pulses are often of good strength, and much benefit will be derived from the cold affusion and ice-cap to the shaven scalp, from an enema to clear out the bowels, and in the young and robust, from the application of from two to six leeches to the temples. A nurse ought to be in constant attendance at the bedside, to prevent the patient getting up and doing himself injury. Blisters I have never found of the slightest service in such cases; while alcohol or opium alone, in place of procuring sleep, would probably increase the delirium. If the above measures fail to quiet the patient, or induce sleep, the remedy which often acts like a charm is opium, in combination with antimony, in the manner first recommended by Dr. Graves.[y] It ought to be given without delay, for the longer this form of delirium lasts, the greater is the danger to the patient. The following prescription may be ordered:—

R. Liq. Op. Sed. . . . ℥j.
Ant. Pot. Tart. . . . gr. j. ad gr. ij.
Mist. Camph. . . . ℥vj. Misce.
Sumat coch. mag. j. omni horâ donec somnus superven.

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[u] Corrigan, 1853, p. 23. [x] Ibid. p. 24.
[y] Graves, 1836 and 1848, i. 207.

It is difficult to say how the action of the opium is modified by the tartar emetic; but that it is modified is shown by numberless instances of the above combination at once inducing sleep, where opium alone has failed. The antimony seems to exercise a tranquillizing influence on the nervous system, so that a much smaller quantity of opium suffices, than when this drug is given by itself. It also induces mild diaphoresis, and thus overcomes one of the main objections to opium, that it tends to lock up the secretions. When the medicine succeeds, the pulse often falls from 10 to 30 beats in the minute, while, at the same time, it is increased in volume: after the second or third dose, the patient often begins to perspire and falls into a good sleep, from which he awakes quiet and more conscious. It is possible that certain other medicines, such as digitalis, aconite, and veratrum viride, which are known to have a remarkably sedative effect on the arterial pulse, may, like antimony, assist the action of opium in the delirium of typhus.[z]

But when the delirium approaches to typhomania or delirium tremens, the radial pulse is usually quick and feeble, the cardiac impulse diminished, and the first sound of the heart more or less inaudible. Here antimony appears to be contra-indicated, and our main reliance must be placed on opium, in combination with alcoholic and other stimulants. Dr. Graves recommended antimony and opium, even in cases of this nature; and, in fact, wherever sleeplessness and delirium of any sort in typhus coexisted: but the cases where I have found the combination of most use are those above mentioned. The amount of stimulants must be regulated by the state of the pulse and heart, and the opium may be prescribed as follows :—

℞. Liq. Op. Sed. . . . ℥ss.
Æth. Chlor. . . . ℥j.
Mist. Camph. . . . ad. ℥iij.

Sumat coch. mag. ij. et rep. coch. mag. j. omni horâ donec somnus superven.

Opium (gr. ½), with camphor (gr. iij.), in a pill, repeated, if necessary, after two hours, is also an excellent combination for inducing sleep under such circumstances. Barrallier has found Scotch paregoric, or the Tinctura Opii Ammoniata of the Edinburgh Pharmacopœa, in doses of fifteen to thirty minims, very useful in these cases.[a] Baron Dupuytren and Dr. Graves[b] found that opiates, in the form of enemata, sometimes succeeded in procuring

[z] Digitalis and veratrum viride have been strongly commended by German and American physicians, as arterial sedatives in the treatment of both Typhus and Enteric Fever.

[a] Barrallier, 1861, p. 377. [b] Graves, 1848, ii. 529.

sleep, after they had been given in vain, in large and repeated doses by the mouth, and I have had more than one opportunity of confirming this observation in the case of typhus. In addition to the opium and stimulants, much benefit will often be derived from warm fomentations to the scalp, and the affusion of tepid water on the head.

Occasionally, it is difficult to decide whether the opiates ought to be combined with antimony or with stimulants. The face is flushed; the skin is hot and dry; and the patient is restless and excitable, and perhaps attempts to leave his bed; but the cardiac and radial pulse is feeble. In such a case, it may be well to combine the two methods of treatment, carefully watching the effects, and regulating the amount of stimulants on the one hand, and of antimony on the other, according to circumstances. Indeed, always when the antimonial treatment is adopted, the patient should be seen by the medical man at least three or four times in the course of the day. The symptoms may change and require a corresponding alteration of treatment.

But there are certain contra-indications to the use of opium in typhus. It must not be given when urgent dyspnœa and lividity of the countenance, betoken extensive pulmonary disease, defective arterialization of the blood, and venous congestion of the brain. It must not be given when there is any tendency to stupor, and it must be discontinued when any such tendency manifests itself. Lastly, it must not be given if there be marked contraction of the pupil, and especially if this contraction approaches to what has been designated *the pinhole pupil*, a condition which may co-exist with violent excitement and wakefulness as well as with stupor. Many years ago, Dr. Graves proposed the use of belladonna as a sedative and soporific in such cases, inferring from its action on the pupil, that it was much less likely than opium to aggravate the injurious effects of the typhus-poison upon the brain.[c] This inference has been strengthened by the observations of Mr. Benjamin Bell,[d] on the antagonistic therapeutic effects of atropia and morphia. I can confirm Dr. Graves' observation as to the occasional utility of belladonna in such cases. The manner in which he prescribed it was as follows:—

R. Ext. Belladon	. . .	gr. j.
Ext. Hyoscy	. . .	gr. vj.
Pil. Hydr.	. . . .	℈j: Misce Div. in Pil. vj.

Sumat i. 3â q. q. horâ.

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[c] GRAVES, 1838

[d] *Edin. Med. Journ.* July, 1858, iv. 1.

Or, ℞ Ext. Belladon. . . . gr. j.
Pulv. Moschi . . . gr. x.
Mist. Acaciæ . .
Syrup. Aurantii. . . a. a. ʒij.
Mist. Camph. . . . ℥ss Misce.
Ft. haust. 6â q.q. horâ repetend.

Henbane may be tried, when opium is contra-indicated; but, to be of any use, it must be given in large doses. Two drachms may be given at once, and one drachm repeated every third hour.

The internal administration of chloroform, in half-drachm doses, has been recommended by Drs. Gordon and Corrigan,[d] as an occasional substitute for opium in cases of typhus and delirium tremens, where sleeplessness is combined with great restlessness, nervous agitation and delirium. Here is Dr. Corrigan's prescription:—

℞ Chloroform. . . . ʒv.
Pulv. Glycyrrh. . . . ʒv.
Mist. Camph. . . . ℥ix. ss. Misce.
Sumat cochlear. mag. ij. 2â q.q. horâ.

Chloroform inhalation, Dr. Corrigan found to be useless in procuring sleep, and to be attended with some danger. He mentions two cases of delirium tremens, where it proved fatal.

Where there is nervous excitement going along with great debility, and characterized by low muttering delirium, tremors, subsultus, carphology, feeble pulse, and inaudible first sound of the heart, musk and camphor are remedies of great value. Of late years, these medicines have fallen into unmerited neglect, probably owing to the great expense of the one, and to the other not being prescribed in sufficiently large doses. Gerhard tells us that he found camphor one of the most useful and powerful remedies in the Philadelphia epidemic of typhus, in 1836. He gave it in emulsion in doses of five grains every two hours, and in enema in doses of a scruple. "The immediate effect was the 'lessening of the subsultus and tremors, and sometimes the dimi-'nution of delirium. In some cases, we possessed a complete 'control over the subsultus, which was immediately checked by a 'camphor injection.'[e] Huss speaks in the highest terms of both musk and camphor, under the circumstances in question. Barrallier also testifies to their great utility in the delirium tremens of typhus.[f] Graves was in the habit of combining musk and camphor with tartar emetic and opium, in cases where there was subsultus tendinum, in addition to the usual symptoms of cerebral excitement. In one case, given in his lectures, where there was likewise

[d] Gordon and Corrigan, 1854. [e] Gerhard, 1837, xx. 320.
[f] Barrallier, 1861, p. 292.

complete sleeplessness, he prescribed a draught every two hours, containing half-a-grain of tartar emetic, ten grains of musk, five grains of camphor, and ten drops of laudanum. After taking three doses, the patient fell into a quiet sleep, and awoke quite rational.[g]

A slight amount of *drowsiness* is the natural mode of termination of typhus, and requires no treatment; but if any difficulty be experienced in rousing the patient, there is danger of the stupor passing into profound and fatal coma. As already stated, this stupor is independent of any anatomical lesion of the brain or its membranes, but is probably due to the circulation in the blood of the products of the retrograde metamorphosis of tissue. Accordingly, the treatment which suggests itself is to promote elimination, more especially by the kidneys, to improve the condition of the blood, to rouse the patient by stimulants applied to the external surface, while at the same time we support the action of the heart. I have reason to believe, that a dangerous degree of stupor is often prevented, by the early adoption of the treatment already recommended. This treatment is still applicable when stupor is present. It is in this condition, that I have often observed the greatest benefit from a strong infusion of coffee, a small cupful of which may be ordered every three or four hours. At the same time, it is well to employ measures which have a derivant action on the kidneys; such as dry cupping, and mustard poultices to the loins, followed by the 'wet compress,'[h] particularly when the presence of albumen or blood in the urine points to a hyperæmic condition of the kidneys, or when the urine is scanty or suppressed. The bowels are to be opened by a purgative or by a turpentine enema, and the action of the skin is to be encouraged by frequent tepid sponging. If the skin be dry, the warm bath, the hot-air bath, and packing in a hot, wet blanket, deserve a trial.

An attempt should also be made to rouse the patient by stimulants to the external surface. For this purpose, blisters to the scalp, or nape, are often most efficacious. Painting with strong acetum cantharidis, is much preferable to the ordinary blistering plaster, which takes effect slowly, and is apt to be torn off by the patient. The *liquor vesicatorius* prepared by Messrs. Bullen and

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[g] GRAVES, 1848, i. 185.

[h] Wet compresses are favourite remedies with hydropathists; but they ought not, on that account, to be discarded by the regular practitioner. They are often of great utility in relieving hyperæmia of the kidneys. Thick flannel folded two or three times is to be wrung out of hot water, passed round the loins, and covered with a piece of mackintosh or oiled cloth, retained in its place by a bandage or towel.

Burt has been used for many years at the London Fever Hospital, and is stronger, and more certain and rapid in its action, than the ordinary preparations. It ought not to be applied to the occiput, which is subjected to pressure. If the blister fail to rouse the patient, sinapisms may be applied to the inside of the thighs, the soles of the feet, or the epigastrium. The cold affusion has been recommended as a stimulant in cases of great stupor, provided there be considerable elevation of temperature, and little irritability of the nervous system. Dr. Armitage ascertained, by careful observation, that the first effect of this treatment was to diminish the temperature, and the frequency of the pulse and respirations, and to moisten the tongue, while the stupor diminished, and sometimes disappeared entirely during the affusion.[i] 'The douche,' says Dr. Todd, 'sometimes acts like a charm; it is most applicable 'to cases, in which a lethargic state supervenes early, and before 'there is great exhaustion.'[k]

The action of the heart is to be supported by alcoholic and other stimulants, according to the instructions already laid down, additional caution being required in their administration, when the urine contains blood or albumen.

In all cases of cerebral oppression, particular attention must be paid to the state of the bladder. The practitioner must not be satisfied with the nurses' report, that the patient has passed water in bed, for a small quantity often dribbles away and makes a great show, when the bladder is enormously distended. The hypogastric region should be examined two or three times daily by manipulation and percussion, and if there be the slightest doubt, the catheter must be introduced. Dr. Corrigan records a case, where violent convulsions, followed by coma, resulted from inattention to the state of the bladder in a case of fever under the care of a homœopath. Dr. Corrigan was called in and drew off the urine; the patient recovered, but suffered from cystitis for more than a year.[l]

Two other remedies have lately been recommended, on good authority, for the coma of typhus, viz.:—Valerian and Phosphorus. I have had no experience of either. The essential oil of valerian was given by Barrallier in 172 cases of typhus, characterized by stupor and coma, and its effects are said to have been almost marvellous. 'Des individus plongés dans une profonde somno-'lence, dont rien ne pouvait les tirer, insensibles à tout ce qui se 'passait autour d'eux, après avoir pris le matin l'essence de

[i] Armitage, 1852, p. 55. [k] Todd, 1860, p. 160. [l] Corrigan, 1853, p. 42.

'valériane, étaient le soir reveillés, répondaient aux questions qu' 'on leur addressait; et ce changement était si imprévu, si étonnant, 'que plusieurs fois, j'ai entendu les personnes qui suivaient mes 'visites prononcer le mot de *résurrection*.' The remedy was successful in 135 of the 172 cases; unsuccessful in 24; and the results were doubtful in 13. About one minim in a little syrup and water was ordered every half-hour, until five or eight minims had been taken.[m]

Phosphorus is highly praised by Huss in cases of extreme torpor and prostration; 'when the patient lies upon his back, quiet, 'without any delirium, indifferent, and not easily roused; when 'the pulse does not exceed 100, and is small and feeble; when 'the first sound of the heart, though audible, is feeble and short, 'and the respiration slow and unimpeded, and when the tempera-'ture does not exceed 101° Fahr.' It is not to be given when there is any delirium. It is given dissolved in almond oil, in doses of $\frac{1}{12}$ of a grain every two or three hours.[n]

6. In every case of typhus, we must be constantly on the outlook for *complications*, the varied character of which has been already pointed out. In their treatment, we must be guided by general principles, and by the symptoms in the individual case, never forgetting, that the patient is labouring under a disease characterized by a remarkable tendency to nervous prostration and depression of the heart's action, and forbidding the employment of depleting or lowering measures.

Pulmonary Complications are the most common:—bronchitis and pulmonary hypostasis, and, in rarer cases, pneumonia and pleurisy.

Bronchitis. In the milder cases, where the affection is limited to the larger tubes, it will be sufficient to add from five to ten minims of the Vinum Ipecacuanhæ to each dose of the acid mixture, or to give night and morning, five grains of the compound conium or ipecachuan and squill pill of the London Pharmacopœia, or three to five grains of Dover's powder, either alone, or in combination with two grains of James's powder. At the same time, a sinapism or turpentine stupe is to be applied to the chest once or twice daily, with wet compresses in the intervals. Wet compresses are strongly recommended by Huss;[o] a single or double layer of flannel moistened in warm water is laid over the front of the chest, or passed round it, and then covered with gutta percha, or waterproof sheeting. In all cases of typhus, if there be the slightest cough or quickness of breathing, the chest ought to be examined daily, and when any bronchial râles are heard, if recourse be had

[m] Barrallier, 1861, pp 168, 376. [n] Huss, 1855, p. 178. [o] Ibid. pp. 138, 158.

at once to the measures now recommended, we may hope to avert the more severe forms of the affection, which are apt to supervene with great rapidity, and too often to resist all treatment.

In the more severe forms of bronchitis, the treatment must vary, according to the stage of the disease and the general symptoms. In those exceptional cases, where bronchitis ushers in the attack, or supervenes during the first week, and when the patient is strong and robust, and the pulse of good strength, great benefit is derived from antimony, the judicious administration of which will sometimes prevent the bronchitis passing into the severe forms, which it often assumes in the advanced stage of the fever. It may be prescribed as follows :—

℞. Ant. Pot. Tart. . . . gr. j. ad. gr. ij.
Potass. Nitrat. . . . ℥j.
Tr. Camph. Co. . . . ʒiij.
Mist. Amygdal. . . . ℥v. ss. Misce.
Sumat coch. mag. 3â q.q. horâ.

The patient must be carefully watched, so that the antimony be not pushed too far. But the cases are few, in which the stage of the disease, or the state of the pulse and heart, will justify the use of antimony. Frequently, the circulation is not only enfeebled, but the lividity of the lips and face, the rapid breathing and inability to cough, the existence of moist râles all over the chest, and of dulness on percussion over the dependent parts of the lungs, indicate both extensive bronchitis, and hypostatic engorgement. Under such circumstances, antimony would only hasten the fatal termination. The pulmonary engorgement is of a passive character, and dependent on want of power in the heart, while the danger consists in the air-passages being blocked up by accumulated secretion, consequent on paralysis of the muscles of expiration. Alcohol and wine must be given freely, while recourse is had to stimulant expectorants, and diuretics, such as senega, ethers, squill, and ammonia, and the nitrate, bitartrate, or acetate of potash.

℞. Potass. Nitrat. . . gr. v.
Sp. Aeth. Nit. . . ʒss.
Tinct. Scillæ. . . ♏xv.
Decoct. Senegæ . . ℥j. Misce.
Fiat. haust. 3â. q. q. horâ sumend.

Or,

℞. Sp. Ammon. Aromat. . . ʒss.
Ætheris Chlorici . . ♏x.
Tinct. Scillæ . . . ♏xv.
Decocti Senegæ . . . ℥j. Misce.
Fiat haust. 3â q.q. horâ sumend.

Dry cupping is often of service; but to do good, it must be prac-

tised extensively over the chest, and be frequently repeated. Counter-irritation, and wet compresses in the intervals ought likewise to be employed. Blisters are recommended by some practitioners; but the turpentine stupe and sinapisms are preferable, as they can be often repeated, and are not liable, like blisters, to degenerate into troublesome sores. If blisters be used, they must not be applied to the back, nor left on too long.

In extreme cases, when the tubes are filled with secretion, the face livid, and the patient has not the strength to cough, or whenever the above treatment fails, recourse should be had to turpentine internally. This remedy was, I believe, first recommended by Huss,[p] who speaks of it as one of the greatest treasures in modern medicine; and certainly, its effects in the bronchitis of adynamic fevers, are sometimes marvellous. It ought to be given in doses of from ten to twenty minims, with fifteen to thirty minims of chloric or sulphuric ether, and half a drachm of spiritus junip. comp., in mistura acaciæ, mistura amygdalæ, or yelk of egg. The dose may be repeated every two hours at first, until the desired effect be produced. After a few doses, the patient often begins to cough, and expectorate large quantities of viscid mucus, with great relief to the respiratory symptoms. The quantity of urine is likewise increased. I have never known strangury produced.

Creasote is another remedy which I have sometimes found to be of great service in such cases, as well as in adynamic bronchitis independent of typhus; and, next to turpentine, deserves a trial. It may be prescribed thus:—

℞. Creasoti . . . .
Acid Acetic. . . . a. a. ℥viij.
Sp. Aeth. Co. . . .
Syrup. . . . . a. a. ʒiv.
Aquæ . . . . . ℥vij Misce.
Sumat coch. mag. ij 2â vel 3â q. q. horâ.

An emetic of mustard is said to act sometimes like a charm in cases of this sort, by promoting copious expectoration, and allowing free ingress of air into the bronchial tubes, so as to save the patient from impending suffocation.[q]

Pneumonia in typhus is usually preceded and accompanied by more or less bronchitis, and requires, for the most part, similar treatment. The symptoms are of such an adynamic character, that depletion and antimony are out of the question. The latter is only permissible under circumstances, similar to those where it has

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[p] Huss, 1855, p. 162; see also Lyons, 1861, p. 170.
[q] Lyons, 1861, p. 169.

been recommended in bronchitis. Professor Strohl, of Strasbourg, has lately obtained excellent results by treating pneumonia with acetate of lead ;[r] and, from frequent trials, I am convinced that it is often a valuable remedy, both in simple pneumonia, and in that which complicates typhus. Similar testimony to its efficacy is borne by Dr. Joseph Bell, of Glasgow,[s] Two or three grains, with or without opium, according to circumstances, may be given every four hours. But, in all cases of pneumonia in typhus, if the circulation be feeble, or if lividity and coldness of the surface point to non-aëration of the blood, recourse must be had to stimulants, expectorants, turpentine, and counter-irritation, as recommended for bronchitis.

In cases where pneumonia or pulmonary hypostasis comes on suddenly, and spreads rapidly over both lungs, so as to threaten suffocation, the propriety of abstracting blood by cupping, or even from the arm, so as to relieve the internal engorgement, has been entertained. In one or two cases of this sort, I have taken a few ounces of blood from the arm, administering stimulants at the same time freely, to keep up the heart's action, but the results have not been such as to justify the repetition of the practice.

When pneumonia passes into *gangrene*, the treatment should consist in nitro-muriatic acid, quinine, and a liberal supply of stimulants and nourishment, together with chlorinated mouth-washes, and the inhalation of tar-vapour or chlorine ; but the case is almost hopeless.

Pleuritic effusions are rare complications or sequelæ of typhus, but, when they occur, are to be met by counter-irritation, painting iodine over the chest, diuretics, iodide of potassium and tonics. When there is reason to believe that the effusion is purulent, the best treatment is quinine and acids.

Care must be taken, not to confound '*cerebral respiration*' (see page 182) with the dyspnœa resulting from pulmonary complications. Cerebral respiration is usually the precursor of coma, and is best treated by a blister to the scalp, evacuation of the bowels, and the remedies already recommended for stupor and coma.

Although when *convulsions* occur in typhus, treatment is seldom of much avail, the case is not altogether hopeless. A blister is to be applied to the shaven scalp, and the bowels made to act freely by means of a turpentine enema, or by one or two drops of Croton oil

[r] *Gaz. des Hôp.* Fév. 28, 1861 ; *Med. Times & Gaz.* Jan 19, 1861.
[s] Bell, 1860, vol. ix, p. 55.

given by the mouth. Saline diuretics ought also to be given, especially if the urine be scanty and suppressed. Dry cupping over the kidneys, and the hot air or vapour bath, are likewise indicated. Alcohol ought not to be given in large quantity, unless the pulse be extremely feeble. In every case, the state of the bladder must be looked to.

For the *muscular or neuralgic pains*, which occasionally occur during convalescence, recourse must be had to quinine and opiates, anodyne liniment, of aconite and opium, etc. Barrallier strongly recommends the inhalation of chloroform.[t]

Hyperæsthesia of the integuments is sometimes relieved by tepid sponging. If this fail, opium may be given internally, provided there be no contra-indication. Barrallier observed great relief from the internal administration of chloroform, in doses of from 10 to 25 minims every hour for four hours.[u]

In cases of *partial palsy* following typhus, the mineral tonics, and a generous diet, with malt liquor and the cold shower-bath, usually effect a speedy cure. In obstinate cases, recourse may be had to galvanism, and small doses of nux vomica or strychnia. Where incontinence of urine persists after fever, the best remedy is the tincture of the muriate of iron; a blister may also be applied over the sacrum, and in the female, immediate relief will often be derived from cauterizing the orifice of the urethra with nitrate of silver.

A generous diet and tonics are also called for in cases of *mental imbecility* and *mania*. In the latter case, it may be necessary occasionally to subject the patient to temporary confinement in an asylum.

Vomiting in the first week of typhus is often arrested by an emetic and by opening the bowels. At a later stage, if there be any tenderness over the stomach, the bowels ought to be cleared out by rhubarb and blue pill, and sinapisms applied to the epigastrium. If the vomiting persist, the acid treatment is to be temporarily suspended, and ice, lime-water, bismuth and magnesia, or some effervescing mixture substituted. Vomiting, occurring when the patient is very prostrate, is sometimes checked by the exhibition of brandy and soda-water or champagne.

Diarrhœa is to be treated with astringents and enemata of starch and opium. Towards the termination of the disease, it may be due to paralysis of the bowels, and then steel and stimulants are indicated.

In cases of *dysentery*, much benefit will be derived from a com-

[t] Barrallier, 1861, p. 173. [u] Ibid. p. 176.

bination of Dover's powder, hydrargyrum c. creta, and ipecachuan, with a dose of astringent mixture, after each motion of the bowels.

Extreme *tympanitis* will often be relieved by a turpentine, assafœtida, or peppermint enema, followed by stimulants, and turpentine stupes to the abdomen. If these measures fail, turpentine ought to be given internally.

℞ Spirit Terebinth.
Æth. Sulph. . . . a. a. ℥xv.
Aq. Menth. pip. . . ʒvij.
Mist. Acaciæ . . . ʒj. Misce.
Ft. haust. 4tâ q. q. horâ sumend.

The same remedies are applicable in cases of urgent *hiccup*, attended by abdominal derangement. More commonly, severe hiccup has a cerebral origin, and must be treated like other cerebral symptoms, with stimulants, ether, camphor, musk, etc., and blisters to the scalp; but, too often, all remedies are unavailing. In several cases, I have observed marked relief from sucking small pieces of ice. Vinegar, in doses of a drachm, has long enjoyed a good repute for the treatment of hiccup under all circumstances, and was strongly commended, forty years ago, for the hiccup of typhus.[x]

In all severe or protracted cases of typhus, the back ought to be examined daily, and means adopted to prevent undue pressure on those parts where *bed-sores* are apt to form, especially the sacrum and hips. The annular air-cushion is an excellent expedient for protecting the sacrum, and when practicable, the patient ought to be laid on a water-bed, spring-bed, or strap-bed.[y] As soon as the slightest redness is discoverable, the parts should be carefully washed with a lotion of camphorated spirit, and painted twice daily with a mixture of collodion and castor-oil, or with the white of an egg beaten up with an equal quantity of rectified spirit, or with a solution of gutta-percha in chloroform. I prefer the last, which is made by dissolving one drachm of sheet gutta-percha in one fluid ounce of pure chloroform. The effect of all these applications, is to stimulate the cutaneous capillaries, and to form a protecting film over the surface. When bed-sores have formed, stimulating poultices ought to be applied until the sloughs separate. An excellent application under such circumstances, is composed of two parts of castor-oil, and one of balsam of Peru, spread on pieces of lint, which are laid on the sore and covered with a linseed poultice, to be changed three or four times a day. Yeast, carrot and

[x] STOKER, 1815. [y] An excellent strap-bed has been invented by Dr. Corrigan (see CORRIGAN, 1853, p. 84).

charcoal poultices, or a few drops of turpentine on the ordinary linseed poultice, are also very useful. To correct fetor, the parts are to be washed each time that they are dressed, with a lotion of chlorinated soda,[z] or of Condy's fluid.[a] After the sloughs have separated, the sores are to be dressed with some stimulating lotion, and if they be slow in healing, or exhibit a tendency to spread, they must be painted daily with a strong solution of nitrate of silver (℈j. ad ℥j.), or touched occasionally with nitric acid, while poultices or water dressings are applied in the intervals.

When the feet are cold, local warmth is to be applied by warm water bottles, and bags of hot sand or bran. As soon as *gangrene* of the extremities threatens, the livid part, and the space for some distance beyond, are to be painted with a strong solution of nitrate of silver, and the limb enveloped in compresses steeped in tincture of camphor, or moistened with a few drops of turpentine, and covered with cotton wool. After gangrene has commenced, the same treatment is applicable as for bed-sores; amputation may be necessary occasionally, after the line of demarcation has formed.

In gangrene of the mouth, it is particularly desirable to limit the disease as soon as possible, by the application of strong nitric acid. Poultices are to be applied externally, and the mouth frequently washed out with chlorinated lotions, or Condy's fluid.

Sloughing, and ulceration of the cornea, are best prevented by covering the eyes with wet compresses, whenever the patient lies constantly with them open. When ulcers have formed, they ought to be touched repeatedly with solid nitrate of silver.

Sloughing in any part of the body, indicates a low state of the system, and calls for large quantities of stimulants, quinine, the mineral acids, and other tonics. As soon as the primary fever has ceased, malt liquors, and abundance of nourishment, in a digestible form, ought to be allowed. Opium is usually required to relieve pain and procure sleep.

Erysipelas is best treated by stimulants and by the muriated tincture of iron and chloric ether, (fifteen minims of the former, and ten of the latter, every three hours), and by fomenting the part with a lotion of acetate of lead and opium (plumb. diacet. et. pulv. op. a.a. gr. iv. ad ℥j.), or painting it with a strong solution of nitrate of silver (℈j. ad ℥j). In erysipelas of the face, we must be on our guard against a similar condition of the pharynx or larynx; and when either of these parts becomes affected, the fauces, back of the pharynx, and the entrance to the larynx, ought to be freely

[z] Liq. Sod. Chlorin. ℥iv., Aq. ℥xjss.

[a] Liq. Pot. Permang, ℥iij, Aq. ℥ixss.

painted with the nitrate of silver solution, or with the muriated tincture of iron, diluted with an equal volume of water. Steam and vinegar exhalations are to be employed, while sinapisms and fomentations are applied externally. When the patient is unable to swallow, brandy, beef-tea, ether, and quinine, ought to be given by the rectum, or when practicable, introduced by a long tube into the stomach. When suffocation is imminent from obstruction of the rima glottidis, tracheotomy holds out the only—although very slight—chance of saving life.

In the *Phlegmasia alba* of the lower extremity, the limb is to be kept elevated by pillows, so that the foot is considerably above the level of the trunk, and strict rest in the recumbent posture enjoined. The entire limb is to be fomented with decoction of poppy-heads, or with the acetate of lead and opium lotion, and outside the oiled silk, a flannel roller is to be applied from the toes to the hip, so as to keep up gentle pressure, and maintain the temperature. Corrigan's 'bandage fomentation,' is an excellent application in such cases. 'Tear up flannel into yard lengths, of 'the usual bandage-width, and, having steeped them for a moment 'in hot decoction of poppy-heads and camomile, make them into 'so many rollers, and squeezing out the superfluous moisture, roll 'them successively round the limb, until they are laid on, three or 'four folds thick, over the extent desired. Then take similar rolls 'of dry flannel, and apply them in like manner, three or four 'folds thick over the first applied and moist rollers.'[b] This application is to be renewed twice or three times a day. If a hard, painful cord be felt in the situation of the femoral vein, it may be advisable to apply three or four leeches over the vessel, or if leeches be contra-indicated by the condition of the patient, strips of lint, smeared with equal parts of belladonna and mercurial ointment, may be laid across the course of the vessel, but neither the leeches nor the ointment must interfere with assiduous fomentation. For some time after the swelling has disappeared, the limb ought to be kept warm and supported by external pressure.

Diffuse cellular inflammation requires similar local treatment to that just described, with free incisions as soon as matter has formed. In *pyæmia*, with purulent deposits in the joints, anodyne fomentations must be applied to the affected parts. In both complications, the internal treatment must be one of support—stimulants and tonics, more especially quinine and the mineral acids, with nutritious food, and opium to relieve pain and procure sleep.

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[b] CORRIGAN, 1853, p. 89.

For *inflammatory swellings* in the parotid region, and elsewhere, the internal treatment must be conducted on the same principles as in gangrene, erysipelas, and pyæmia. Fomentation with the lead and opium lotion, and poultices, are to be employed locally, and as soon as matter has formed, it ought to be evacuated by free incisions. I have employed leeches in the early stage, in several instances, without any benefit. I have also treated a considerable number of cases by repeated painting with strong solution of nitrate of silver, or with tincture of iodine. In several instances, the swelling has appeared to recede after the former application.

For *anasarca* of the lower extremities, tonics, more especially steel, with nutritious diet, and bandages externally, should be prescribed.

Treatment during Convalescence.

As soon as the fever ceases, most patients convalesce rapidly, unless there be some complication; and the chief duties of the physician consist in preventing premature exertion and exposure to cold, and in checking the inordinate appetite. Although there is probably no acute disease in which the appetite returns more speedily, and may be gratified with greater impunity, it is well to restrict the diet, for the first two or three days of convalescence, to animal soups and farinaceous articles, with milk and eggs. On the third or fourth day, if the tongue be clean and moist, the pulse slow and the rash gone, a piece of boiled white fish or chicken, or the lean part of a mutton chop, may be allowed. As soon as convalescence is established, porter or ale ought to be substituted for the wine and brandy, as they are more fitted for promoting the transformation of food, and, at the same time, furnish nutriment themselves, in the form of gluten and sugar.

The bowels are usually costive, and are to be kept open by mild laxatives and enemata. The mineral acids, with bark, quinine, and iron, may be given as tonics, and are particularly called for when the pulse is abnormally slow, in which case, also, the patient should be cautioned against assuming the erect posture too soon, as sudden and fatal syncope has sometimes been the result. Opiates and henbane may be required to produce sleep; and in every case great benefit will be derived from a change of residence and exercise in the open air. When convalescence is retarded by complications, recourse must be had to the measures already mentioned.

CHAPTER III.

Relapsing or Famine Fever.

Section I.—Definition.

A CONTAGIOUS disease which is chiefly met with in the form of an epidemic, during seasons of scarcity and famine. Its symptoms are: a very abrupt invasion marked by rigors or chilliness; quick, full, and often bounding pulse; white moist tongue, sometimes becoming dry and brownish; tenderness at the epigastrium; vomiting, and often jaundice; enlarged liver and spleen; constipation; skin very hot and dry; no characteristic eruption; high-coloured urine; severe headache, and pains in the back and limbs; restlessness, and occasionally acute delirium; an abrupt cessation of all these symptoms, with free perspiration, about the fifth or seventh day;—after a complete apyretic interval, during which the patient may get up and walk about), an abrupt relapse on the fourteenth day from the first commencement, running a similar course to the first attack, and terminating on or about the third day of the relapse; sometimes a second, or even a fifth relapse;—mortality small, but occasionally death from sudden syncope, or from suppression of urine and coma;—after death, no specific lesion, but usually enlargement of liver and spleen.

Section II.—Nomenclature.

1.—*Names derived from its duration and peculiar course.*

A Five Days' Fever with Relapses (*Rutty*, 1770); Short Fever, Five Days' Fever (*var.*, 1817-19); Five, or Seven days' Fever (*Wardell, etc.*, 1843, *Irish Writers*, 1847); Remittent Fever (*Craigie*, 1843, *Purefoy*, 1853); Relapsing Fever (*Paterson, Steele, etc.*, 1847; *Jenner*, 1849; *Lyons and Anderson*, 1861); Typhus recurrens (*Hirsch*, 1859); Das recurrirende Fieber (*German Writers*); Fièvre à rechute (*French Writers*).

2.—*Names derived from its Prevalence in Epidemics.*

The Epidemic Fever (*auct. var.*); Epidemic Fever of Edinr., 1817 (*Welsh*, 1819); Epidemic Fever of Ireland *pro parte* (*Barker and Cheyne*, 1821); Scotch Epidemic of 1843 (*Alison, Wardell, R. Cormack, Jackson,*

Henderson, H. Douglas, D. Smith, Craigie, etc.); Epidemic Remittent Fever (*Mackenzie*, 1843); the Silesian Fever of 1847 (*Brit. and For. Med. Ch. Rev., July*, 1851).

3,—*Derived from the supposed Inflammatory Nature of the Pyrexia.*

Dynamic or Inflammatory Fever (*Stoker*, 1835; *and Dublin Journal*, 1848); Synocha (*Cullen*, 1769; *Christison*, 1840 *and* 1858); Relapsing Synocha (*Seaton Reid*, 1848).[e]

4.—*Derived from the common occurrence of Jaundice as a Symptom.*

Yellow Fever (*Graves and Stokes*, 1826; *Arrott*, 1843); Bilious Relapsing Fever (*Steele*, 1848); Gastro-hepatic Fever (*Ritchie*, 1855). Has also been designated Bilious Remittent Fever, Remitting Icteric Fever, Biliary Fever, and Bilious Typhoid Fever.

5.—*Derived from its connection with Famine.*

Famine Fever (*Stoker*, 1826, *and Irish Writers generally*); Armentyphus (*German Writers*, 1848); Die Hungerpest (*Grœvell's Notizen*, 1848).

6.—*Other Synonyms.*

Fever of the New Constitution (*O'Brien*, 1828); Miliary Fever (*Ormerod*, 1848; *Watson*, 1848); Typhinia (*Farr*, 1859),

Section III.—Historical Account of Relapsing Fever.

RELAPSING Fever, like typhus, is not a new disease. Dr. Spittal has shown that Hippocrates described a fever as prevailing, upwards of two thousand years ago, in the island of Thasus, off the coast of Thrace, which resembled it very closely in most of its characters, including an intermission of five or seven days between the febrile attacks, jaundice, epistaxis, tendency to miscarry, &c.[d]

In the accounts of many epidemics of typhus, mention is made of relapses, which in some instances probably referred to relapsing fever, as this fever prevails often as an epidemic, in conjunction with typhus. Strother, in describing the fever epidemic in London, in 1729, speaks of frequent relapses;[e] and Lind, in his account of the contagious typhus of the fleet, observes: 'in the 'fevers concerning which we are treating, the patients are very 'subject to relapses.'[f]

The earliest mention, however, of relapsing fever, on which reliance can be placed, occurs in Rutty's 'Chronological History 'of the diseases of Dublin.'[g] Speaking of the year 1739, he says:

[e] Relapsing Fever probably constituted one of the varieties of the 'Inflammatory Fever,' or 'Synocha' of the writers of last century; more recently, it has often been considered a variety of Typhus.

[d] Spittal, 1844, p. 177; *Hippocrat. Op.* Syd. Soc. ed. i. 389.

[e] Strother, 1729, p. 121. [f] Lind, 1763, p. 63. [g] Rutty, 1770.

'The latter part of July, and the months of August, September, 'and October, were infested with a fever, which was very frequent 'during this period, not unlike that of the autumn of the preceding 'year; with which compare also the years 1741, 1745, 1748. It 'was attended with an intense pain in the head. It terminated 'sometimes in four, for the most part in five or six days, some-'times in nine, and commonly in a critical sweat: it was far from 'being mortal. I was assured of seventy of the poorer sort at the 'same time in this fever, abandoned to the use of whey and God's 'good providence, who all recovered. The crisis, however, was very 'imperfect, for they were subject to relapses, even sometimes to 'the third time. In some, there succeeded pains in the limbs." Again, at page 90, after speaking of the typhus of 1741, he says: 'Through the three summer months, there was frequent here and 'there a fever, altogether without the malignity attending the 'former, of six or seven days' duration, terminating in a critical 'sweat; but in this the patients were subject to a relapse, even to 'a third or fourth time, and yet recovered.' Huxham described frequent relapses in the fever prevalent at Plymouth, in this same year.[h]

Relapsing fever also appears to have been observed by Dr. John Clark, at Newcastle in 1777.[i]

During the epidemic of 1797-1801, many cases of relapsing fever were observed. 'Certain it is,' remarked Barker and Cheyne, 'that the fever in 1801 very generally terminated on the 'fifth or seventh day by perspiration, and that the disease was then 'very liable to recur; and that the poor were the chief sufferers 'by it.'[k]

There is evidence of the occasional occurrence of relapsing fever, during the first sixteen years of this century, in Ireland and elsewhere,[l] while the next great epidemic of fever (1817-19), was chiefly composed of it. It is needless to recapitulate the circumstances under which this epidemic originated, or the extent of its prevalence (see page 36). Typhus and relapsing fever were then regarded as modifications of one disease, and according to Dr. Christison, 'there was a general impression that the relapsing fever 'could produce the common typhus.' Hence, it is not surprising that the records of the epidemic do not show the period, at which each fever was most prevalent. But the circumstance, that the rate of mortality increased at many places with the advance of the epidemic, makes it probable that the proportion of typhus to

[h] Huxham, 1752. [i] Clark, 1780, pp. 36, 132. [k] Barker & Cheyne, 1821, i. 20.
[l] Ibid. 213.

relapsing cases was greater towards the close of the epidemic, than at its commencement. Thus, of 28,514 cases of fever admitted into the Dublin Hospitals, from September, 1817, to November, 1818, inclusive, 1242 died, or 1 in 23; while of 9,419 cases admitted during the first six months of 1819, 525 died, or 1 in $17\frac{2}{3}$.[m] In the Cork Street Fever Hospital, of 7,613 cases admitted in 1818, there died 256, or 1 in 30; but of 3,920 cases admitted in 1819, 226 or 1 in $17\frac{1}{3}$.[n]

Again, of 1741 cases admitted into the Waterford Fever Hospital, during the first nine months of 1818, only 51, or 1 in $34\frac{1}{51}$ died, while of 2,050 cases admitted during the last three months of 1818, and the first three months of 1819, there died 122, or 1 in $16\frac{9}{61}$.[o] From Dec. 16th, 1817, to June 16th, 1818, there were admitted into the Fever Hospital, at Ennis, 206 cases, of whom 10, or 1 in $20\frac{3}{5}$ died, while from June 16th, to Dec. 16th, 1818, 22 died out of 281 cases, or 1 in $12\frac{1}{2}$.[p] In Aberdeen we are told, that in January, 1819, towards the end of the epidemic, the disease assumed a worse aspect, and the number of fatal cases increased.[q]

It would not be difficult to multiply these results, and, in fact, an increase in the rate of mortality, with the advance of the epidemic, was all but universal.[r]

After 1819, relapsing fever seems to have almost disappeared until the subsequent epidemic of 1826, which consisted of both typhus and relapsing fever, but in which the proportion of typhus was greater than in the preceding epidemic. Now, for the first time, a distinction was drawn between the two fevers, and there is conclusive evidence that the proportion of relapsing cases was greatest at the commencement of the epidemic, and progressively diminished as the epidemic advanced. Dr. O'Brien, who published an account of the epidemic, as it appeared at Dublin, states that, at the commencement, there were 'two fevers, the ordinary 'typhus, or fever of the old constitution,' which was very fatal, and 'a fever of the new constitution,' lasting only a few days, and seldom fatal, but frequently relapsing. At first, he says, most of the cases were of the latter form, but as the epidemic advanced, the proportion of relapsing cases greatly decreased.[s] This statement is confirmed by comparing the rate of mortality of the epidemic at different stages of its progress. Thus, of 8,607 cases

[m] HARTY, 1820, 6th Table of Appendix. [n] Ibid. p. 40.
[o] BARKER & CHEYNE, 1821, ii. 48. [p] Ibid. 108. [q] HARTY, 1820, p. 115.
[r] It is well known that in epidemics of pure typhus the mortality is greatest at the commencement. (See page 219). [s] O'BRIEN, 1828.

admitted into the Dublin Fever Hospital, from May to December, 1826, only 249 died, or 1 in $34\frac{7}{12}$; whereas, of 3,658 cases admitted from from January to May, 202 died, or 1 in $19\frac{42}{101}$. A similar observation was made by Alison, with regard to the epidemic in Edinburgh. He states, that the symptoms generally were more asthenic than in the epidemic of 1817-19, and that this was more especially the case in 1827 than in 1826.[t] It also appears from Alison's memoir, that the rate of mortality from fever in the Royal Infirmary was greater in 1827 than in 1826.

From 1828 to 1842, relapsing fever may be said to have disappeared from Britain. It formed no component part of those extensive outbreaks of fever at Glasgow and Edinburgh in 1831-2, and 1840-1, or of the more general epidemic of 1836-38. Its cessation was so complete, that when it again broke out, in 1843, it was regarded by many as a new disease. In Ireland, its disappearance was perhaps not equally complete, but even there, little or no mention was made, during this period, of a fever presenting its peculiar characters.

Towards the end of 1842, and in 1843, appeared that remarkable epidemic in Scotland, and, to a less extent, in England, which has already been described (page 47). This epidemic resembled that of 1817-19, in consisting mostly of relapsing fever. True typhus, however, was not absent, and increased in prevalence with the advance of the epidemic. This circumstance is clearly brought out by the returns of the Glasgow Royal Infirmary, where, as in Edinburgh, the two fevers were now recognized as distinct diseases, and the numbers of each carefully recorded. Thus:

	Relapsing Fever.	Typhus.	
In 1843, were admitted,	2,871	and 142,	or $20\frac{1}{4}$ R. F. to 1 Typhus
In 1844, „	„	432 and 711,	or 1 „ to $1\frac{31}{48}$ „
In 1845, „	„	37 and 266,	or 1 „ to $7\frac{7}{37}$[u] „

The following, also, is the rate of mortality from fever, during the same period, in the Edinburgh Infirmary, showing a considerable increase towards the close of the epidemic :—

Oct. 1, 1842, to July 1, 1843, 817 admissions, and 6·85 p.c. died.
July 1, 1843, to Oct. 1, 1844, 4,642 „ and 7·77 p.c. „
Oct. 1, 1844, to Oct. 1, 1845, 679 „ and 11·34 p.c.[x] „

Dr. Wardell's Tables also show that, of 330 patients in the Edinburgh Infirmary, in October, 1843, only 10, or 1 in 33, had the

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[t] Alison, 1827.
[u] McGhie, 1855, p. 161. [x] *Statistical Reports of the Hospital.*

eruption of typhus, which was present in 24 of 450 patients (or 1 in $18\frac{3}{4}$) in the hospital during the following January.[y]

Dr. Rose Cormack, who, in December, 1843, published a monograph on the epidemic, as observed in Edinburgh, thus wrote:—'As the season advanced, all the cases have been more characterized by depression and general typhoid symptoms. The cases of *Continued Fever*, with and without measly eruption, are becoming more and more common in Edinburgh, and also in Glasgow, as Dr. Weir, of the Infirmary there, informs me.'[z] In the *Medical Gazette*, for April, 1849, the same writer observes:—'Towards the close of the epidemic (of 1843), the ordinary Edinburgh typhus, with measly eruption, began to rage.'

In the London Fever Hospital, 'the peculiar typhus eruption' was noted in only 1 of 61 cases admitted in January, 1844, but in 22 of 39 cases admitted in August; again, of 111 cases admitted in December, 1843, only 3 (or 1 in 37) died, whereas of 39 cases admitted in August, 1844, 11 (or 1 in $3\frac{6}{11}$) died. The reports also state, that relapses occurred in almost all the cases admitted in the latter part of 1843, but were rare in 1844.

After the epidemic of 1843, a few cases of relapsing fever continued to be observed, both in Ireland and Britain, until the end of 1846. The epidemic of 1847-8 presented a greater proportion of typhus cases, and in this respect, bore very much the same relation to the epidemic of 1843, that the epidemic of 1826 bore to that of 1817-19. The greater preponderance of relapsing fever at the commencement of the epidemic, was a matter of general observation. Thus, Dr. Steele, in his report of the cases admitted into the Royal Infirmary, at Glasgow, observes:—'It will be seen, by reference to Table XIII., that the two diseases kept steadily advancing, somewhat in an inverse ratio. At the beginning of the year, the cases of relapsing fever averaged about three-quarters of the whole admissions. The disease advanced, though very gradually, till the month of July, after which the number began to decline, and at present (April, 1848,) they form but a small proportion of the cases under treatment. The number of typhus cases admitted in January, 1847, was so low as 66. The admissions increased rapidly till July, when they out-numbered those of the rival epidemic. After this period, typhus cases began to decline very slowly, at the same time, always keeping ahead of the relapsing cases; so that, at the close of the year, the former averaged about two-thirds of the whole fever cases under

[y] Wardell, 1846, xxxvii. 229, 774.

[z] Cormack, 1843, p. 107.

'treatment.'[b] The following are the actual numbers of admissions of each fever into the Glasgow Royal Infirmary:—

In 1846 . .	777	Relapsing Fever	.	500	Typhus.
In 1847 . .	2,333	„	„	. 2,399	„
In 1848 . .	513	„	„	. 980	„
In 1849 . .	168	„	„	. 342[c]	„

Mr. James Paterson, speaking of the Barony Fever Hospital, at Glasgow, which was opened for eleven months, from Aug. 5, 1847, remarks:—'The relative proportion of the two principal forms of 'fever, varied much at different periods of the Hospital's history. 'At its opening, the number of cases of fever, with relapse, doubled 'that of the typhus cases. At the close of the year, they were 'nearly equal, and during, and after, February, the number of the 'typhus cases doubled that of the relapse cases.'[d]

The same sequence of events was noticed at Edinburgh. From statistics of the epidemic, published by Dr. R. Paterson, it appears that, from May 1, 1847, to January 31, 1848, 589 cases of relapsing fever, and 422 cases of typhus, came under treatment; whereas, during the two months of February and March, 1848, the numbers were 58 of relapsing fever, and 73 of typhus.[e] Again, the Official Statistical Tables of the Infirmary, show that, from October 1, 1848, to October 1, 1849, there were admitted 203 cases of relapsing fever, and 349 of typhus; whereas, from October, 1849, to October, 1850, there were only 25 cases of relapsing fever, to 468 cases of typhus.

Similar observations were made in London, by Dr. Ormerod[f] and others. Of 64 cases of 'fever,' admitted into the London Fever Hospital, in April, 1847, at the commencement of the epidemic, only 1 died; whereas, of 104 cases admitted in December, 12 (or 1 in $8\frac{2}{3}$) died; and of 707 cases admitted in the year 1848, 148 (or 1 in $4\frac{4}{5}$) died. On the whole, however, cases of relapsing fever were few in London, in proportion to typhus. The cases in the London Fever Hospital did not exceed 100.

The Irish records of this epidemic render it probable, that the same order of events took place in that country. Although the accounts are less clear, inasmuch as few Irish physicians recognized the distinctions between the different forms of fever, the following extract from Dr. Kennedy's account of the epidemic in Dublin is to the point: 'Cases of genuine typhus were through the whole 'epidemic very rare. Occasional cases did occur, and these

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[b] Steele, 1848, p. 166. [c] McGhie, 1855, p. 161. [d] J. Paterson, 1848, p. 361.
[e] R. Paterson, 1848, p. 397. [f] Ormerod, 1848, p. 217.

'became more numerous with the advance of the epidemic.'[g] Throughout the epidemic, the proportion of true typhus cases appears to have been much less in Ireland than in Scotland, and in Scotland than in England.

The years 1846 and 1847, were marked by severe famine, not only in this country, but in some parts of the continent, more particularly in the Prussian province of Upper Silesia, and in some other parts of Germany. There, an epidemic broke out, which was the precise counterpart of that in the British Isles. The investigations of many accurate observers, such as Virchow, Dümmler, and Suchanek,[h] leave no doubt that this epidemic consisted partly of relapsing fever, and partly of typhus. It commenced in Upper Silesia, where the effects of the famine were felt most severely, and where the condition of the inhabitants singularly resembled that of the Irish. The following paragraph is extracted from a review of the epidemic by an English writer:

'The province of Upper Silesia is a dependency of Prussia. It 'is inhabited, however, not by Saxons, but by a race of Poles, who 'have been severed from their nation for 700 years, and yet have 'preserved their language, their religion, and their unwillingness 'to labour, although they have lost the inventive genius, and the 'chivalrous spirit of their parent stock. Separated thus from 'Prussia by differences of blood, of religion, and of language, the 'utmost efforts of that enlightened country have failed to teach 'them Saxon industry, or to give them Saxon comfort. The 'schoolmasters, who have been sent among them, have learned 'Polish, but have not taught German; the Protestant teachers 'have only excited in them a more fanatic zeal for their Catholic 'priests: the profound literature of Germany awakened in them 'no response; and amidst the clash and tumult of modern pro-'gress, they remain silent and unmoved in their antique isolation. 'Like the Irish, the potato is their staple article of food, to which 'they add butter-milk and sauerkraut. Their dwellings are the 'prototypes of the Irish cabins, and in the smallest and dirtiest 'huts, persons of all ages and sexes are crowded together. Nor 'does the parallel to Ireland end here. The relations between 'landlord and tenant appear to be on as false a footing, as those 'which exist in Ireland, only that here a still more oppressive state 'of servitude may be found. The aristocracy, also, as in Ireland, 'adopt a system of absenteeism, and spend in Berlin or Vienna,

[g] *Irish Report*, 1848, vii. p. 54: also, viii. p. 67; H. KENNEDY, 1860, p. 217.
[h] See *Bibliography*, 1849.

'the small portion of wealth which the labour of their miserable 'dependants creates. The Silesians, like the Irish, are excessively 'intemperate.'[i]

Since the epidemic of 1847-8, relapsing fever has been gradually disappearing, and for the last seven or eight years, not one case has been observed in the hospitals of Edinburgh, Glasgow, or London. Dr. W. T. Gairdner has not seen or heard of a single case at Edinburgh, since 1855.[k] In London, there was a considerable increase of relapsing fever in 1851, when it was found that the patients were almost exclusively Irish, many of whom had been but a short time in London, and that all were in a state of extreme destitution. This increase of relapsing fever occurred at a time when typhus was comparatively rare; but gradually, the number of relapsing cases diminished, and that of typhus increased. (See Table XVIII., and Diagram I.) In Glasgow, there was also an increase of relapsing fever in 1851, followed by a great increase of typhus, as the relapsing cases gradually disappeared. Both in London and in Glasgow, I have reason to believe, that not a single case of relapsing fever has been observed since 1855.[l] The following table shows that the admissions for typhus, and relapsing fever, into the London Fever Hospital, and the Glasgow Infirmary, since 1847.

TABLE XVIII.

Years.	London Fever Hosp.		Glasgow Royal Infirmary.	
	Typhus.	Relap. Fever.	Relap. Fev.	Typhus.
1848	786	13	513	980
1849	155	29	168	342
1850	130	32	174	382
1851	68	256	255	919
1852	204	88	192	1293
1853	408	16	72	1551
1854	337	5	68	760
1855	342	1	22	385
1856	1062	—	—	385
1857	274	—	—	314
1858	15	—	—	175
1859	48	—	—	175
1860	25	—	—	229
1861	86	—	—	509
1862	1500(?)	—	—	

With regard to Ireland, Dr. Purefoy described a modified form

Review, 1851, p. 28. [k] GAIRDNER, 1859, p. 48, and 1862, No. 2, p. 158.
[l] *Reports of Glasgow Royal Infirmary.*

of relapsing fever, in which the relapses were uncertain and irregular, as very common in Tipperary, so late as 1853,[m] and I am informed by Dr. M'Evers, that in the same year, it was 'very rife' at Cork. Both Dr. M'Evers, and Dr. Lyons, however, write to me that in Ireland, true relapsing fever has of late years been a rare disease.

In the present epidemic in London, the cases have been exclusively typhus. I have not seen or heard of a single instance of relapsing fever.

From the above remarks, and from the observations formerly made in the historical account of typhus, the following conclusions are arrived at:—

1. Relapsing fever is an epidemic disease, in a stricter sense, than even typhus. It may disappear entirely for years from those places, where at other times it rages most fiercely.

2. Epidemics of relapsing fever have usually co-existed with epidemics of typhus, and have always appeared under circumstances of distress or famine.

3. In mixed epidemics, the relative proportion of typhus and relapsing cases has varied at different times and places; but, as a rule, the proportion of relapsing cases has been much greater at the commencement than towards the close of the epidemic, and with the advance of the epidemic, typhus has taken the place of relapsing fever.

Section IV.—Geographical Distribution.

IRELAND and Britain are the countries where epidemics of relapsing fever have been chiefly observed. In England, the fever has never been so prevalent as in Scotland, and with the exception of 1843, it has usually been more prevalent in Ireland than in Scotland. Indeed, it is not difficult to show that a very large proportion of the patients attacked with relapsing fever in Britain are of Irish birth or extraction. Of the 441 cases of relapsing fever admitted into the London Fever Hospital since 1847, the birth-place was noted in 420, as follows:—

Table XIX.

Natives of London	83, or 19·76	per cent.
„ of rest of England	50, or 11·9	„
„ of Scotland	2, or ·47	„
„ of Ireland	281, or 66·9	„
Foreigners	4, or ·95	„
	420 99·98	

[m] Purefoy, 1853.

It appears, then, that of the total number of cases, more than two-thirds were natives of Ireland. Adopting the census of 1851 (see page 57) as a basis of information, concerning the birthplaces of all the inhabitants of London, it follows that during the period above mentioned, there were admitted into the London Fever Hospital—1 in every 386, of the Irish inhabitants; 1 in every 8,351 of Foreigners; 1 in every 15,200 of the Scotch inhabitants; 1 in every 16,465 of the English inhabitants. Moreover, a large proportion of the patients born in London or the rest of England, were the children of Irish parents, or were obviously, from their names, of Irish extraction. Many also of the Irish patients had only recently arrived from Ireland; of 250 Irish cases, whose length of residence in London was ascertained, 20, or 8 per cent., had left Ireland within three months; 36, or 14·4 per cent., within six months; and 81, or 32·4 per cent., within a year.

The Irish have long been the reputed importers of epidemic fever into Britain, and it is a question what fever they have introduced. It has already been shown, with tolerable certainty, that, so far as London is concerned, they do not introduce typhus, and now it seems equally certain that the fever which they introduce is relapsing fever. The contrast between the Table given at page 56, and the results in the case of relapsing fever, leaves no doubt on the point. The epidemics whose importation has been attributed to the Irish, have been, for the most part, mixed epidemics of typhus and relapsing fever, and the number of Irish cases has been greatest at the commencement of the epidemic, when the proportion of relapsing cases was largest (see page 299).

Take, for example, the epidemic of 1847. All accounts agree in stating that it did not commence in Glasgow, Liverpool, and other towns, until after the immigration of large numbers of destitute Irish.[n] According to Dr. R. Paterson, 'at the commence-
'ment of the epidemic in Edinburgh, almost every case admitted
'into the Infirmary was from Ireland, and for nearly three
'months they continued so.' Large numbers had come direct from Ireland. With the increase in the proportion of cases of true typhus, the proportion of Irish patients diminished, and that of the Scotch increased (see page 49).[o] It follows, that the cases of relapsing fever, of which this epidemic was at first mainly composed (see page 296), were, for the most part, Irish. Similar observations were made in London: of the patients admitted into

[n] See page 48.

[o] R. Paterson, 1848; see also Orr, 1847, p. 374.

the Fever Hospital at the commencement of the epidemic, the majority were suffering from relapsing fever, (see page 296), and 'a considerable proportion were poor Irish, who had not been 'in London many days, and who had reached the Metropolis with 'fever on them, or destitute of food and clothing, and in an 'extreme state of exhaustion.'[p] Dr. Ormerod, from his experience at St. Bartholomew's, stated that the cases of relapsing fever, in 1847, were 'mostly Irish newly arrived in London,' and added:— 'At first the residents still continued to suffer from the better 'known form of the disease in all its severity (typhus), whereas 'the newly-arrived Irish had mild relapsing (miliary) fever.'[q]

With this epidemic it is important to contrast that which is at present prevailing in London. The fever has been entirely typhus; not one case of relapsing fever has occurred; and, at the same time, the proportion of Irish has been comparatively small (see page 57).

It must not, however, be supposed, that relapsing fever is peculiar to the Irish. The Scotch epidemic of 1843, originated in Scotland, and scarcely, if at all, implicated Ireland. Of 150 patients at Edinburgh, observed by Wardell, at an early stage of the epidemic, only 25 were natives of Ireland, and they had caught the disease by lodging in houses or localities where it prevailed. As the epidemic advanced, the proportion of Irish increased.[r] (See page 47).

Again, in 1847, an epidemic of relapsing fever, identical with that observed in Britain, prevailed in the Silesian provinces of Prussia and Austria;[s] and, in the summer of 1855, after the hardships and privations of the preceding winter, it is said to have been 'pretty common among the British troops in the 'Crimea.'[t] With these exceptions, I am not aware that relapsing fever has been met with in any other part of the continent of Europe.

The observations of Dubois,[u] Austin Flint,[v] and others, leave no doubt that relapsing fever was observed at New York and Buffalo, and in other parts of North America, during 1847 and 1848; but all the cases were traceable to Irish immigrants, and there are no grounds for believing that the disease is ever indigenous in America. In India, and indeed in all tropical countries, relapsing fever is unknown.

Professor Griesinger, of Tubingen, when in England, in 1859,

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[p] *Report for* 1847, p. 11. [q] Ormerod, 1848, p. 217. [r] Wardell, 1846, xxxvii. 229. [s] See page 297. [t] Lyons, 1861, p. 106. [u] Dubois, 1848. [v] Flint, 1852.

informed me, that the fever which he had observed at Damietta, in Egypt, and described as '*Das biliose Typhoide*,'[x] was identical with the relapsing fever of this country. But on referring to his original account of this disease, in Virchow's Archives for 1853,[y] I think that there can be no doubt that the Egyptian disease is very different from relapsing fever, and that it is a malarious remittent fever, perhaps closely allied, as Griesinger thought at the time he wrote, to yellow fever. The sudden fall of the pulse from 120 to 75, observed in many cases, was not attended by that general improvement in the symptoms, which occurs during the apyretic interval of relapsing fever. Indeed, this fall in the pulse, was usually the immediate forerunner of severe typhoid symptoms, and was attributed by Griesinger to an admixture of bile with the blood, as it was only observed in cases where there was jaundice. There was nothing like a distinct crisis, on or about the seventh day, with a relapse about the fourteenth. The mortality was very much greater than that of relapsing fever; 25 of 132 cases, or 19 per cent., died. Lastly, quinine was found to be of signal service, whereas in relapsing fever, this remedy is known to be useless.

Section V.—Etiology of Relapsing Fever.

A.—Predisposing Causes.

1. *Sex.*

OF 441 cases admitted into the London Fever Hospital, since the commencement of 1848, 233 were males, and 208 females. The following tabular statement, collected from different records, shows that the number of males treated in different hospitals for relapsing fever, has, on the whole, somewhat exceeded that of the females. The difference, however, has not been great; and, moreover, the excess has not always been in favour of the males, so that it is improbable that sex, in itself, predisposes to relapsing fever.

	M.	F.	Total.
London Fever Hospital	233	208	441
Wardell[z] (Epid. 1843. Edin.)	159	171	330
Cormack[a] (,, ,,)	75	92	167
H. Douglas[b] (,, ,,)	122	93	215
W. Robertson[c] (Epid. 1847-8, Edin.)	358	231	589
R. Paterson[d] (,, ,,)	325	314	639
Edin. Infirmary Reports, 1848-9,	110	93	203
Steele,[e] (Epid. 1847-8, Glasgow)	1159	1174	2333
	2541	2376	4917

[x] Griesinger, 1857, pp. 199, 202.
[y] Ibid. 1853.
[z] Wardell, 1846.
[a] Cormack, 1843.
[b] H. Douglas, 1845.
[c] Robertson, 1848.
[d] R. Paterson, 1848.
[e] Steele, 1848.

The remarks made on this subject, under the head of typhus, are equally applicable here. (See page 61).

2. Age.

The following Table shows the ages of 437 cases admitted into the London Fever Hospital, since the commencement of 1848.

TABLE XX.

Age.	No. of Cases.			Per centage at each period of life.
	M.	F.	M. & F.	
Under 5 years	2	2	4	·91
From 5 to 10 years ...	18	14	32	7·32
„ 10 to 15 „ ..	41	22	63	14·42
„ 15 to 20 „ ...	54	38	92	21·05
„ 20 to 25 „ ...	40	36	76	17·39
„ 25 to 30 „ ...	16	21	37	8·46
„ 30 to 35 „ ..	18	19	37	8·46
„ 35 to 40 „ ...	8	11	19	4·34
„ 40 to 45 „ ...	16	24	40	9·15
„ 45 to 50 „ ..	4	4	8	1·83
„ 50 to 55 „ ..	8	7	15	3·43
„ 55 to 60 „ ...	5	2	7	1·6
„ 60 to 65 „ ...	—	5	5	1·14
„ 65 to 70 „ ...	—	1	1	·23
„ 70 to 75 „ ...	1	—	1	·23
„ 75 to 80 „ ...	—	—	—	—
Above 80 years	—	—	—	—
Age doubtful	2	2	4	—
Total, omitting doubtful cases	231	206	437	99·96
Mean Age	22·98	26·01	24·41	—

The youngest case was a female aged 2, and the oldest, a man aged 74.

From this, it would appear that relapsing fever attacks all ages, but that the proportion of the young to the aged is greater than in the case of typhus. Nearly one half of the cases of typhus were found to be upwards of 30, and nearly one eighth upwards of 50; whereas, only one-third of the relapsing cases are above 30, and only one-fifteenth above 50. Again, of typhus less than 1 in 6, but of relapsing fever, more than 1 in 4½ are under 15 years of age. The contrast between the ages of the two fevers is apparent from the following tabular comparison :—

	Per cent. of Typhus cases.	Per cent. of Relapsing cases.
Under 10 years there were	5·78	8·23
,, 15 ,, ,,	16·3	22·65
From 15 to 25 years,,	30·12	38·44
25 years and upwards	53·58	38·9
30 ,, ,,	43·66	30·43
40 ,, ,,	26·47	17·62
50 ,, ,,	11·92	6·63
60 ,, ,,	4·68	1·6

In relapsing fever, as in typhus, females of an advanced age seem more liable to be affected than males. Thus, at the London Fever Hospital, the mean age of the females has in every year exceeded that of the males, and

Out of a total of 441 cases,	233	were males;	208	females.
Of cases below 25 years	155	,,	112	,,
Of cases above 25 ,,	76	,,	94	,,

Another point worthy of notice, and which was also observed in the case of typhus, is the great absolute increase of cases between the ages of 40 and 45, beyond that in the preceding lustrum. This increase was not, as in typhus, restricted to females.

These results agree with most of the statistics of Relapsing Fever, which have been published. Of 203 cases admitted into the Edinburgh Infirmary during the years 1848-9, 45 or 1 in $4\frac{1}{2}$ were under 15 years of age; 50, or one-fourth, above 30; and only 9, or less than 1 in 22, above 50.[f] Of 215 cases under Halliday Douglas in 1843, 77 were under 20, 135 under 30, 80 above 30, and 28 above 50.[g] Lastly, of 2,333 cases in Steele's report of the Glasgow epidemic of 1847, 302, or 1 in $7\frac{2}{3}$, were under 15; 795, or about one-third, were above 30; and 153, or one-fifteenth, were above 50.[h]

3.—*Months and Season of Year.*

Table XXI. shews the number of cases of Relapsing Fever admitted in each month into the London Fever Hospital, as compared with the number of admissions into other Hospitals.

The largest number of cases have been admitted into the London Fever Hospital during the summer and autumn months; but the undue preponderance in these months was produced by the cases of one year, as shown by the second column of the Table. Relapsing Fever is truly an epidemic disease, and the season of the year seems to have little influence on its prevalence. In one epidemic, the largest number of cases occurs during one season; in another

[f] *Statistical Reports.* [g] DOUGLAS, 1855. [h] STEELE, 1848.

epidemic, during a different season. In Edinburgh in 1843, the epidemic was at its height during the autumn and winter: the Glasgow epidemic of 1847 was at its climax in spring and summer. The Edinburgh epidemic of 1843 commenced in January or February, the Dublin epidemic of 1826, in May; the Leith epidemic of 1843, in September; and the Glasgow epidemic of 1843, in December, 1842. Epidemics of Relapsing Fever appear then to commence, progress, and decline, quite irrespectively of the season of the year. Relapsing Fever differs, on the one hand, from Enteric Fever, in not being always most prevalent in autumn; and from Typhus, in not being usually most prevalent during and towards the end of winter.

TABLE XXI.

Months and Seasons.	Lond. Fever Hospital.[i]		Edin. Royal Infirmary.		Glasgow Royal Infirmary in 1847.[k]
	From 1848 to 1862.	In 1851.	In 1843.[j]	In 1844.[j]	
January	22	1	—	465	192
February	27	4	74	300	181
March	17	4	83	256	265
April	46	17	96	93	226
May	46	30	133	50	226
June.....	43	30	161		222
July	31	18	251		239
August.....	44	37	392		198
September ...	23	15	531		182
October	56	38	638		162
November ...	49	34	586		133
December.....	37	28	544		107
Spring	90	25	253		672
Summer	120	78	545		687
Autumn	123	90	1561		542
Winter	108	63	1595		432

4. *Occupation.*

There is no proof that any occupation, in itself, predisposes to relapsing fever. A large proportion, however, of the cases admitted into the London Fever Hospital have been costermongers, street musicians, or beggars, a circumstance probably due to the precarious nature of their subsistence. It has been a common observation at all times and places, that a large proportion of the

[i] See note l. p. 65. [j] ALISON, 1843. [k] STEELE, 1848.

persons attacked with relapsing fever are vagrants, with no fixed residence. (See Table VII., page 68).

5. *Recent Residence in an Infected Locality.*

The annexed Table shows the length of residence in London of all the cases of relapsing fever admitted into the London Fever Hospital since 1847, in which the circumstance was noted.

Table XXII.

Less than 3 months	34	or	8·95 per cent.
„ 6 months	56	„	14·73 „
„ 1 year	102	„	26·84 „
„ 2 years	161	„	42·37 „
„ 10 years	259	„	68·16 „
More than 10 years, but not for life	38	„	10· „
For entire life	83	„	21·84 „
Total	380	„	100·00 „

From these figures, it might seem that recent residence in London does predispose strongly to relapsing fever. Of 380 cases, one-eleventh had not resided in London more than three months, and many, only a few days; more than one-seventh had not resided more than six months; and considerably upwards of one quarter, not more than a year; not many more than one-fifth had resided in London all their lives.

This result, however, is not due to any local cause in constant operation, for, during the last seven years, not a case of relapsing fever has occurred in London. It is partly due to the circumstance that a large number of the persons attacked with relapsing fever are vagrants or out of employment, who, after wandering over the country in search of work or food, arrive destitute and exhausted in the crowded habitations of large towns, where the disease is already prevalent. But both in London and in other places, it has been a common observation, that not a few of the patients have been actually ill at the time of their arrival. It has been already shown that a large proportion of the patients came from Ireland. Of 34 patients who had resided in London less than three months, 20 came from Ireland; of 56, who had resided less than six months, 36 came from Ireland; and out of 102 cases, who had been in London less than a year, 81 came from Ireland. It may be reasonably inferred, that, in many instances, the disease is contracted in Ireland; while, in others, it may be a matter for future enquiry, whether it has not been generated by the priva-

tions and exhaustion to which the patients have been subjected during their journey. I am inclined to think that the latter supposition is not altogether impossible. (See pages 316-7).

6. *Over-crowding and Destitution.*

Relapsing fever, being, like typhus, communicable from the sick to the healthy, over-crowding, of course, favours its propagation. Accordingly, relapsing fever is found to prevail chiefly in the most crowded localities of large cities, inhabited by the poorest of the population. Of 441 cases admitted into the London Fever Hospital, more than one-half came from the central division, or most crowded part of the Metropolis, and considerably more than one-third from the single district of Holborn. (See Table IX., page 72).

The subject of over-crowding and destitution, in relation to the origin of relapsing fever, will be again referred to in greater detail.

The remarks already made, as to the effects of cold and wet, intemperance, bodily and mental fatigue, depression of spirits, etc., as predisposing to typhus, apply with equal force to relapsing fever. (See pages 66 and 69).

B. Exciting Causes.

1. *Contagion.*[1]

All observers, with the exception of Craigie and Virchow, have believed relapsing fever to be contagious. Craigie, writing in the midst of the Edinburgh epidemic of 1843, when the disease, for the first time, was beginning to be regarded as distinct from typhus, and before sufficient evidence had been collected as to its contagious character, stated, that the belief that it was contagious was a 'presumption rather than a well-founded inference.'[m] Virchow, whose experience of the disease was limited to a fortnight's visit to Silesia, during the epidemic of 1847, came to the conclusion that the disease was not contagious, but was the result of local causes endemic in Silesia.[n] All the medical men, however, practising in Silesia, believed it to be contagious.[o]

That there is a specific poison in relapsing fever, communicable from the sick to the healthy, is proved beyond doubt, by similar evidence to what has been adduced in the case of typhus.

a. When relapsing fever commences in a house or district, it often spreads with great rapidity. Thirty cases have been admitted into

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[1] See note, p. 79. [m] Craigie, 1843, p. 417. [n] Virchow, 1849, p. 263. [o] Ib. p. 254.

the London Fever Hospital from the same house, and 66 cases from the same court, within a few months; and similar observations have been made at all times and places, when the disease has been epidemic.

b. The prevalence of relapsing fever in single houses or in limited districts, is in direct proportion to the degree of intercourse between the healthy and the sick. This was observed to be the case at Glasgow, and in other parts of Scotland, in 1843. In many houses inhabited by several families, when the disease appeared in one apartment, it first attacked all its occupants, and then spread to the rooms adjacent, and afterwards sought its victims in the rooms on the same floor, in the order of vicinity and intercourse. The two following instances, recorded by Mr. Reid, of Glasgow,[p] are to the point, while, at the same time, they demonstrate the importation of the disease into localities before exempt.

The first has reference to the introduction and propagation of the fever at the Dalmarnock colliery, in 1843. This was a large tenement, standing alone and surrounded on every side by open fields. It consisted of three stories, entered by three separate stairs, and inhabited by forty different families. In May, an Irish family removed to a single apartment on the uppermost flat, the youngest child being at the time sick of the fever. On the second of June, the father sickened, and, in succession, the whole family. The disease then spread from house to house, and in the space of two months attacked twenty-two persons on this flat, the other inhabitants of the building being all this time exempt. The absence of the fever before the arrival of the infected family, and its subsequent propagation, first in the infected family, and afterwards among those only in closest communication with them, are facts quite inexplicable on the supposition of a local origin, and indeed in any other manner than on the supposition of contagion.

Secondly, 'the disease was introduced by a person from a 'neighbouring village into a house of two apartments, situated 'in Mile-end, and containing within its narrow walls eleven human 'beings. All of these were attacked, and every one relapsed; but 'in the next house, with a similar entry, and separated only by a 'brick partition, where the occupants were nearly equally numerous, 'and, from their circumstances and habits, equally susceptible, all 'escaped.' Now, if relapsing fever is not contagious, and arises from malaria in the atmosphere, as many have maintained, why was it confined to the one house into which it was introduced, and did not extend to other houses in the immediate vicinity?

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[p] W. Reid, 1843, p. 360.

But, again, most observers testify to the great liability of the attendants on the sick to contract the disease. In 1819, Dr. Welsh, of Edinburgh, wrote thus:—'Since Queensberry-house 'was opened on the 23rd of February, 1818, my friends, Messrs. 'Stephenson and Christison, the matron, two apothecaries in suc- 'cession, the shop-boy, washerwoman, and 38 nurses, have been 'infected; four of the nurses have died. With the exception 'of two or three nurses, who have been but a short time in the 'hospital, I am now the only person in this house, who has not caught 'the disease, within the last eight or ten months.'[q] Dr. Cormack, in his account of the epidemic of 1843, at Edinburgh, observed:— 'Almost all the clerks, and others exposed to the contagion, have 'been seized. Dr. Heude, and his successor, Mr. Reid, in the 'new Fever Hospital; Dr. Bennett, my successor there; Mr. 'Cameron, and his successor, Mr. Balfour, in the adjoining fever 'house, as well as most of the resident and clinical clerks in the 'Royal Infirmary, have gone through severe attacks during the 'past summer and autumn. Hardly any of the nurses, laundry- 'women, or others coming in contact either with the patients or 'their clothes, have escaped; at one time there were eighteen 'nurses off duty from the fever; and, of those who have recently 'been engaged for the first time, or of those who have hitherto 'escaped, one and another is, from time to time, being laid up.'[r]

Similar observations were made at Glasgow and at other Scotch towns, in 1843; and in Silesia, in 1847. It is worthy of notice also, that, in the Scotch hospitals, it was only those nurses and medical attendants, who were brought in contact with cases of relapsing fever, that contracted the disease. The nurses in the surgical wards, and in those medical wards where fever cases were not admitted, escaped. If the fever had been dependent on local causes, all ought to have suffered alike.

c. Persons living in comfortable circumstances, and in localities where the disease is unknown, are attacked on visiting infected persons at a distance. Relapsing fever is a disease peculiar to the destitute, and only attacks persons in easy circumstances, who have had direct communication with the sick. Medical men, living in localities where the disease is unknown, have often been attacked immediately after exposure to the poison. A remarkable illustration is recorded by Wardell. Within a space of five months, in 1843, the resident physician in one of the fever hospitals at Edinburgh had to be re-appointed six different times, five of the

[q] Welsh, 1819, p. 45. [r] Cormack, 1843, p. 115.

gentlemen who held the post, having in succession been attacked by the raging epidemic. All of these gentlemen, had, previous to their attack, resided in different and distant parts of the new town, where the epidemic was scarcely known, yet, as soon as they were exposed to contagion, they at once contracted the distemper.[s]

d. Relapsing fever has often been imported by infected persons into localities before exempt. Certain localities have been observed to become foci for the propagation of the disease, immediately after, but not before, the introduction of infected persons. Two illustrations have already been given on the authority of Mr. Reid, of Glasgow. Many others might be added. In hospitals, it has always been found that the nurses and attendants never contracted relapsing fever, until after the admission of patients suffering from that form of fever. In the hospitals alluded to by Welsh and Cormack, (see page 309), the buildings had not before been used for the treatment of sick persons; in that mentioned by Wardell, none of the workmen, who had been engaged for a long time before in the extensive alterations of the interior, took the fever.[t]

From the foregoing evidence, it is clear that relapsing fever is communicable by the sick to the healthy. So far as our knowledge extends, its infectious principle appears to be governed by the same laws as that of typhus.

1. *The mode of communication* is probably the same as in typhus, that is to say, the poison is conveyed through the air from the sick to the healthy, and actual contact is not necessary.

2. *The distance to which the poison will travel through the atmosphere.* All evidence tends to show, that the remarks made on this subject, under the head of typhus, apply with equal force to relapsing fever. It is only they who are in close communication with the sick, or who visit, or reside in, their badly ventilated dwellings, that suffer. With free ventilation, the disease almost ceases to be communicable. Dr. Cormack states, that in the epidemic of 1843, there were many instances where relapsing fever was imported into houses in the new town of Edinburgh by medical students and others, who had contracted it by visiting the sick, but that he had never known an instance of its spreading in these localities. He also mentions an instance, where a single case of relapsing fever was treated in a general ward; only one of the other patients, a man suffering from epilepsy, contracted the

[s] WARDELL, 1846, xxxvii. 775. [t] Ibid.

fever: this man, and this man only had been in the habit of sitting on the sick man's bed.[u]

3. *Fomites.* The poison of relapsing fever appears to be communicable by clothes. At least, we cannot in any other way account for the fact mentioned by Cormack, of the large number of laundry-women who contracted the fever in the Edinburgh Infirmary, during the epidemic of 1843.[x] These laundry-women had no communication with the sick, except through their clothes and bedding, and their circumstances were not such as to render a spontaneous origin possible.

4. *Length of exposure necessary in order to contract the disease.* If the poison be concentrated, its effects may be manifested at once, but few instances of this nature have been recorded. A medical friend of the author's, visited the Union Workhouse of the City of London, during a period of 1845, when upwards of 100 cases of relapsing fever had been sent from that building to the Fever Hospital. He was attacked on the spot with nausea and headache, and took to bed at once with the fever. When the poison is more dilute, the danger seems to increase with the length of exposure, and, on the whole, a longer exposure appears necessary than in the case of typhus. According to Cormack, very few of the numerous medical officers of the Edinburgh Dispensary in 1843 contracted the disease, in comparison with the number of the medical attendants at the Infirmary. The former were much exposed to the fever in the badly ventilated dwellings of the poor, but were usually with their patients for short periods only, and had constant opportunities for inhaling an uncontaminated atmosphere.

5. *The latent period.* From what has been above stated, it is probable, that the effects of the poison of relapsing fever may occasionally be almost instantaneous; but, on the whole, instances of this description are rare. There are few data for fixing the latent period of relapsing fever with accuracy; but such as exist, make it from four to ten days. Cormack mentions two instances, in one of which the symptoms of the disease commenced between three and four days after the first exposure to the poison, and in the other within five days.[z] Virchow mentions the case of a German physician, who was attacked within nine days after his arrival in Silesia in 1847. The Silesian physicians, however, fixed the latent period at from a fortnight to three weeks.[a]

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[u] Cormack, 1843, p. 116. [x] Ibid. pp. 115, 117. [z] Ibid. p. 117.
[a] Virchow, 1849. p. 262.

6. *Proportion of persons liable to be attacked, on exposure to the poison of relapsing fever.* This is probably smaller than in the case of typhus. During 14½ years, 80 cases of typhus originated in the London Fever Hospital, the number of admissions of typhus during the same period being 4787. One case of typhus, therefore, originated in the institution to every 59 admissions. During the same period, only one case of relapsing fever originated in the hospital, although the number of admissions amounted to 440. This, and other circumstances seem to indicate, that the poison of relapsing fever is feebler in its operations than that of typhus.

7. *Immunity from subsequent attacks.* Contrary to what was found to be the case with typhus, one attack of relapsing fever confers no immunity from subsequent attacks. Welsh tells us, that, in the epidemic of 1817-19, there were several instances of persons having two, and even three, attacks;[b] and Christison observes that during this same epidemic, he experienced no fewer than three separate attacks within fifteen months, in his own person.[c] Wardell[d] and Mackenzie,[e] in the epidemic of 1843, met with several examples of persons having a second attack, after some months; Jenner, from his experience of relapsing fever in London in 1847-50, arrived at the same conclusion;[f] and in the Irish epidemic of 1847, many individuals had a second, or even a third attack, at intervals of a few months.[g]

2. *Spontaneous Generation.*[h]

Although relapsing fever is undoubtedly contagious, it is highly probable that it can be generated *de novo*. A large number of patients are quite unable to trace their illness to contagion. Of 440 cases admitted into the London Fever Hospital since 1847, 171 (or 38·86 per cent.) ascribed their illness to contagion, mostly in consequence of other cases occurring in the same house, while the remainder were not cognizant of any exposure to the poison. Of 213, in which this point was investigated by Douglas, in 150 the disease was referred to contagion, and in 63, no such exposure could be made out.[i] It must also be remembered, that the occurrence of many cases of any disease simultaneously in one house, is no absolute proof that it is contagious. But a stronger argument in favour of a spontaneous generation of relapsing fever, is the fact,

[b] Welsh, 1819, p. 46.
[c] Christison, 1858, p. 583.
[d] Wardell, 1846, xxxvii. 230.
[e] Mackenzie, 1843, p. 226.
[f] Jenner, 1850, xxiii. 119.
[g] *Irish Report*, 1848, viii. 65.
[h] See page 8.
[i] Douglas, 1845.

that after it has been entirely absent for many years, it again breaks out, on each occasion under precisely similar circumstances, and occasionally, as in Scotland in 1843, without any traceable importation. From the fact, that epidemics of typhus and relapsing fever often co-exist, it may be assumed that the conditions under which both diseases originate, are similar, and these conditions may be summed up in two words—destitution and over-crowding. Accordingly, in all accounts of both typhus and relapsing fever, it is stated, that the cases have been confined to the poorest of the population, and, for the most part, to the most crowded localities of large cities. A closer investigation of the etiology of the two diseases, renders it probable, that, while the poison of typhus is generated by overcrowding, and destitution favours its extensive propagation, that of relapsing fever is more intimately connected with, if it be not generated by, destitution, and is propagated by over-crowding.

In the first place, it may be well to demonstrate the intimate connection between relapsing fever and destitution. Of 440 cases admitted into the London Fever Hospital since 1847, 430, or 97·5 per cent. were paid for by the parochial authorities, and totally destitute. Nine of the remaining patients were admitted 'free,' and were also very destitute. Not a single patient had been a servant in a private family, and in only one instance was a fee for admission paid by the patients' friends.[k] A large proportion of the patients, for some time previous to their attack, had been literally starving.

Before the outbreak of the epidemic of relapsing fever in Ireland, in 1817, the inhabitants, owing to a succession of bad harvests and other causes (see p. 38), had for a long time been reduced to *extreme* starvation; and many were reduced to feeding on indigestible articles, such as grass and the roots of trees. Similar observations were made in Silesia, in 1847. Prior to the outbreak of the epidemic, a succession of three bad harvests had reduced the inhabitants to such a state of starvation, that numbers died from this cause alone, and many subsisted on clover, grass, mushrooms, the roots of trees, etc.[1] The state of misery and destitution, under which this same epidemic broke out, in Great Britain and Ireland (where a large proportion of the cases were also at first relapsing fever), is in the memory of all. Speaking of Glasgow, in 1847, Dr. Orr writes: 'The fever hospitals

[k] The reader is referred to the Table under the head of 'Pythogenic Fever;' see also page 74. In another case (not included in the 440), the admission fee was paid; but it is very doubtful, if the case was really relapsing fever.

[1] VIRCHOW, 1849, p. 177.

'were crowded to overflowing with houseless wanderers . . . Many 'poor, starved, destitute, and diseased creatures were brought and 'laid down before the gates of the Infirmary, their relatives, if 'they had any, not knowing what to do with them; and, in nume- 'rous instances, it was destitution and starvation more than fever, 'which was their chief affliction. To destitution, therefore, we 'are principally to look for the cause, which, during the last year 'has filled our fever hospitals to overflowing.'[m] These remarks applied with equal force to every locality in the kingdom, where the epidemic was observed.

But, admitting all these facts, it may be argued, that the famine and the fever are both the results of one common cause—of inclement weather, or of some subtle atmospheric agency. Weather, however, is found to have no influence over the origin or propagation of relapsing fever. It prevails alike in seasons remarkable for the amount of rain (Silesia, 1847), and in seasons remarkable for their drought (Edinbugh, 1843), in unusually hot summers (Edinburgh, 1843), and in the cold of winter (Glasgow, 1842-3, and Leith, 1843-4). Destruction of the crops from any sort of weather seems sufficient to produce it. With regard to any atmospheric agency capable of destroying the fruits of the earth, and, at the same time of inducing relapsing fever, its existence is in the first place a gratuitous assumption, while it is known that relapsing fever may appear quite irrespectively of failures of the crops, and under circumstances where the destitution and misery of the population have, so to speak, an artificial origin. One of the most remarkable epidemics of relapsing fever on record—the Scotch epidemic of 1843—was not preceded by failures of the crops. (See page 47). It did not affect Ireland, but was confined to Scotland, where its connection with destitution was proved by Alison, and many other observers. In 1840, Alison called the attention of the authorities to the deplorable condition of the poor in Scotland, and to the inadequate measures provided by law for their relief.[n] Owing to the construction of railways, which, it is said, attracted numbers of Irish labourers, and caused the inhabitants of the small villages and towns along the lines, to flock into the large towns and to swell their pauper population, and to other causes, the misery and want of the poor, year by year, increased. Between the spring of 1840 and 1843, four public subscriptions, amounting to £20,000, were raised in Edinburgh alone, to relieve their immediate necessities. A charity fund was subscribed in Edinburgh

[m] ORR, 1848, p. 371. [n] ALISON, 1840.

to find employment for the poor, and the coincidence between the progress of the fever, and the cessation of the operations of this fund, were remarkable.

	Men employed by Charity Fund.	No. of Admissions for Fever into Royal Infirmary. [o]
February (1843)	933	74
March	556	83
April	320	96
May	119	113
June	35	161
July	25	251
August	—	392
September	—	531
October	—	638 [o]

During the months of September and October, from thirty to fifty applicants had to be sent away daily from the gates of the Infirmary. The disease was entirely confined to the poor. We are told that some of the medical men in Edinburgh, whose practice lay among the better classes, did not see a single case; while, on the other hand, it was calculated by Alison, that of the destitute poor of Edinburgh, scarcely one escaped. In Glasgow, it is stated, that for two years before the appearance of the fever, the poor had been in an extreme state of privation; and it is added, that the epidemic 'made its appearance, and began to spread, in those 'localities where poverty and wretchedness of every description 'most abounded; and that during the whole season of its preva-'lence, the pauper population were almost its only victims.' [p] In Leith we are told that 'great misery and destitution prevailed 'among the poor.' [q]

Of 1768 cases, collected by Alison,[r] Halliday Douglas,[s] and Murray,[t] 1179, or about two-thirds, were out of employment and utterly destitute at the time of seizure, and many of the remainder had also been out of employment, and only got work a few days before. Moreover, it is important to observe, that the proportion of the very destitute among the patients attacked, diminished as the epidemic advanced. Of 177 patients in the Edinburgh Infirmary, on July 22nd, 127 were out of employment, whereas, on September 30th, this remark applied only to 184 cases out of 330. There are no data for ascertaining the precise proportion, at the very commencement of the epidemic.

Similar observations were made in London, as shown by the

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[o] Alison, (1) 1844. [p] D. Smith, 1844, (2) p. 79. [q] R. Jackson, 1844, p. 418
[r] Alison, (1) 1844. [s] Douglas, 1845. [t] Murray, 1843.

following extract from the Annual Report for 1843 of the Fever Hospital:—'The present epidemic has afforded striking and ex-
'tensive evidence of the close connection between fever and desti-
'tution. A large proportion of the subjects of fever, received into
'the hospital during the past year, were agricultural labourers and
'provincial mechanics (not Irish), who had been induced to leave
'their native counties in search of work, and who, either *on their*
'*road to the Metropolis*, or soon after their arrival in it, were
'seized with the disease. The causes assigned for their illness by
'these poor creatures themselves were various, some stating that
'it was owing to sleeping by the sides of hedges, others to want
'of clothing, many being without stockings, shirts, shoes, or any
'apparel, capable of defending them from the inclemency of the
'weather; while others, and these constituted a very large pro-
'portion of the number, attributed it to want of food, being
'driven, by their intense hunger, to eat raw vegetables, turnips,
'and even rotten apples; and certainly their appearance, in many
'instances, fully corroborated the truth of their representations.'

Having now shown the intimate connection between the origin and progress of relapsing fever and destitution, I proceed to adduce some arguments in favour of the opinion, that in its origin, it is more independent of over-crowding than typhus, and that it is the result of destitution alone.

It is not easy to isolate destitution and starvation from over-crowding. These two conditions almost invariably co-exist. Accordingly, in many of the accounts of relapsing fever, it is stated, that not only were the patients most destitute, but that they inhabited localities which were densely over-crowded.[u] But relapsing fever is found also to prevail where destitution alone could operate, which is seldom, if ever, the case with typhus, except when its origin can be traced to contagion. In Ireland, during great epidemics, it attacks the inhabitants of the country villages and the houseless poor by the way-side, as well as the inmates of the crowded lodging-houses of the large towns. The Scotch epidemic of 1843 did not commence in the large towns, as typhus almost invariably does, but in the country-districts of Fife. In Edinburgh, in 1843, we are informed by Dr. Craigie, that the epidemic prevailed, not only in the crowded localities of the Grass-market, and in the closes of the High-street, the Canongate and the Cowgate, but that 'a number of cases were sent from Musselburgh,

[u] See for example, WARDELL, 1846, xxxvii. 153; R. JACKSON, 1844, p. 418; D. SMITH, 1844 (2), p. 79; PERRY, 1844, p. 85.

'Tranent, Penicuick, Haddington, Dunbar, and similar situations, 'where the population was not dense, and where ventilation was 'excellent.'[x] Mr. Bottomley described an outbreak of relapsing fever among Irish reapers at Croydon, in 1847. They had suffered greatly from privations consequent on the famine, but had not been subjected to over-crowding, for they had been in the habit of sleeping on the roadsides and under hedges. Even on the supposition that the fever, in this instance, was due to contagion imported from Ireland, it is to be observed, that true typhus, whose poison, from all we know of it, is much more active, rarely, if ever, makes its appearance under such circumstances.[y] (See page 86).

But, secondly, it has been typhus, and not relapsing fever, which was observed in the crowded hospitals, ships and prisons, of former days, and which is met with as a consequence of over-crowding in the intervals of great epidemics, when there is no general famine.

Conversely, it is Relapsing Fever, and not Typhus, which has been observed to result more directly from starvation. To the evidence already given on this point, the following statements by Irish observers of the epidemic of 1847, may be added. Dr. Lynch, of Loughrea, reported: 'Most of the cases of fever supervening 'upon the starvation-state, were characterized by repeated re-'lapses, and short febrile attacks. I saw no instances of the 'short relapse-fever amongst the gentry, except in clergymen and 'physicians.[z] Dr. Falkiner, of Kilkullen, reported his experience, in almost the same words.[a]

Fourthly, the voracious appetite often observed during the paroxysms, and peculiar to Relapsing Fever, indicates its more intimate connection with starvation.[b]

Fifthly. The fact, already dwelt on, that, in mixed epidemics of typhus and Relapsing Fever, occurring during seasons of famine, the former fever chiefly prevails at the commencement of the outbreak (p. 299), points to its more intimate connection with destitution. The result of famine has usually been, that the poor have flocked from the country districts, to swell the pauper population of the large towns, which become more crowded the longer the famine lasts. As this crowding increases, the fever, which results from crowding (typhus), is gradually substituted for that which is more immediately the result of destitution.

Lastly, some of the appellations bestowed on Relapsing Fever

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[x] CRAIGIE, 1843, p.417. [y] BOTTOMLEY, 1847.
[z] *Irish Report*, 1848, vii. 393. [a] Ib. viii, 84. [b] See under 'Symptoms.'

in different countries, indicate the popular opinion as to its origin. It is essentially the *Famine Fever* of the British Isles, and the *Hungerpest* of Germany. (See page 291).

From these considerations, the question of identity or non-identity of Typhus and Relapsing Fever naturally arises. That, in their course and symptoms, the two diseases are as distinct as can be, is indisputable; the question which here suggests itself, is, whether the poison (or the circumstances capable of generating it) of the one fever, be, or be not, a modification of the other?

Prior to the epidemic of 1843, Relapsing Fever was regarded as a mild modification of typhus (see page 292). Dr. Henderson, of Edinburgh, in his Clinical Lectures, and afterwards in a paper read before the Medico-Chirurgical Society of that city, on December 6th, 1843,[c] had the merit of first showing that the two diseases were not only very different in their symptoms, but that there was reason to believe that they arose from distinct poisons. His views were confirmed by many other observers, so that since the epidemic of 1843, relapsing fever and typhus have very commonly been regarded as distinct diseases.

The evidence adduced by Dr. Henderson was two-fold; first, that the one fever never communicated the other; and, secondly, that an attack of the one conferred no immunity from an attack of the other.

That the one fever could not communicate the other, was inferred from the circumstance, that examples of the two fevers were never found co-existing in the same house or family. If one of a family had typhus, all the other cases in the same house or family were typhus; if, in one instance, the fever was relapsing fever, it was so in all. From February to September, 1843, Dr. Henderson had seen but 39 cases of typhus; and in 29 of them the histories were carefully investigated. In only 4 could there be the slightest suspicion, that the attack of typhus arose from communication with persons ill of relapsing fever. These 4 cases occurred in houses where relapsing fever was prevailing; but in all the 4 cases it was proved, that the patients had previously been exposed to the contagion of eruptive typhus. In 1849, Dr. Henderson's observations on this point were confirmed by Dr. W. Jenner, who showed that during the three years, 1847-8-9, there had been admitted into the London Fever Hospital, from 2 to 8 cases of typhus from the same house or family, in 57 different instances, making in all 164 cases; and that, in no instance, did a case of relapsing fever come from

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[c] HENDERSON, 1843.

the same house or family as a case of typhus; while during the same period, 2 or more cases of relapsing fever were admitted in many instances from the same house or family, but never associated with typhus.[d]

On the other hand, excellent observers have made observations of an opposite character. Dr. Alison observed, that, in 1843, he had seen at Edinburgh, two cases of typhus, 'with the character-'istic eruption, brought from the same room, in which a succession 'of relapsing cases had occurred at the same time.[e] Dr. David Smith records 5 different instances, which he met with during the same epidemic, at Glasgow, where the two fevers co-existed in the same family.[f] In most of these instances, patients affected with the two fevers, had come from the same bed. Dr. Henry Kennedy, in the epidemic of 1847-8, at Dublin, repeatedly met with cases of both relapsing fever and typhus, occurring in the same room, and amongst members of the same family, often sleeping in the same bed.[g]

The records of the London Fever Hospital, since Dr. Jenner's observations were made, show that the two fevers may occasionally co-exist in the same house or family. They show, moreover, that in certain limited localities, there may at first be nothing but relapsing fever, then relapsing fever and typhus may prevail together, while, still later, there is nothing but typhus. Thus, in the last six months of 1851, there were admitted from Field Lane, in the City of London, into the Fever Hospital, 9 cases of relapsing fever, but none of typhus; in 1852, there were, from the same locality, 14 admissions of relapsing—the last in May, and 24 of typhus—the first in March; and in 1853, there were 16 admissions from the same lane—all typhus. Again, from Tyndall's Buildings, Holborn, there were admitted in 1851, 30 cases of relapsing and one of typhus; in 1852, 10 of relapsing and 12 of typhus. From Pheasant Court, Holborn, there were admitted in 1851, 59 cases of typhus and 3 of relapsing fever; in 1852, 7 of relapsing fever and 5 of typhus; and in 1853, 1 of typhus and none of relapsing fever. From Plum-Tree Court, City, there were admitted, in 1851, 5 of relapsing fever and none of typhus; and in 1852, 3 of relapsing fever and 5 of typhus. In several instances, cases of relapsing and of typhus were brought from the same house, within a few weeks or days of each other. These statements will be better understood by the

[d] JENNER, 1849 (1). [e] ALISON, 1843.
[f] D. SMITH (2), 1844, p. 75.
[g] *Irish Report*, 1848, vii. 53; also, H. KENNEDY, 1860, p. 218.

annexed tabular arrangement, in which the dates are denoted by the names of the months, and the numbers of the houses in each court or lane, by the figures. (R. S. denotes Ragged School, and ? that the number of the house was not noted).

From this, it seems possible, that, at those times when typhus and relapsing fever are both epidemic, it may depend on the precise stage of the epidemic whether we do, or do not, find the two diseases co-existing in the same house or court. Here, in circumscribed localities, there was the same sequence of the two fevers, as was found in studying the history of wide-spread epidemics: at first relapsing fever only, then relapsing fever and typhus together, and, last of all, typhus alone. Whatever be the explanation, the circumstance is most remarkable; but it does not justify the conclusion, that the two fevers are identical. On the supposition, that relapsing fever is generated by destitution; that destitution is the great predisposing cause of typhus; and that typhus is produced by over-crowding, the offspring of destitution; the above is precisely the sequence of events that might be expected. Or, discarding the doctrine of spontaneous generation, it must be admitted, from the facts formerly stated (p. 299), that the foci for the propagation of typhus multiply with the advance of a mixed epidemic, while those of relapsing fever diminish, and that consequently the substitution of one for the other, or their occasional co-existence in a circumscribed locality, does not establish their identity. As far as I know, the statement remains uncontroverted, that in all cases where fever can be proved to have been imported into a locality by a single case, typhus has produced typhus; and relapsing fever, relapsing fever.

The second argument adduced by Dr. Henderson in support of the non-identity of typhus and relapsing fever, was the circumstance, that an attack of one conferred no immunity from an attack of the other. He appealed to nine instances in which the same person contracted the two fevers, within a very short time; and indeed, so general was this observation in 1843, that the managers of the Edinburgh Infirmary made a regulation, that there should be separate wards for typhus and the 'short fever.' It cannot be denied, that such facts have a most important bearing on the question at issue, and that they deserve careful investigation.

Dr. Henderson gives the details of eight of the nine instances to which he alludes. In six, typhus was followed by relapsing fever; in some of the cases, not more than a month intervening between the two attacks; in two, relapsing fever was followed by typhus.[h]

[h] HENDERSON, 1843.

Table XXIII.

Years.	Field Lane.		Spread Eagle Court.		Pheasant Court.		Plum Tree Court.	
	Rel. Fever.	Typhus.	Rel. Fever.	Typhus.	Rel. Fever.	Typhus.	Rel. Fever.	Typhus.
1851.	July, R.S., 10. Aug. 1, 10. Sept, R.S., R.S. Oct., R.S. Nov., ?. Dec., R,S.		Apr., 7, ?, 11, 7, 15. May, 12, ?, 7, ?, 7, 15. June, 15, 9, 9, 9, 11, 1. July, ?, ?, ?, ?, ?. Aug., 7, 8, 11, 8, 10, 10. Oct., 8. Nov., 12.	Sept., 12.	April, 3, 3, 3, 5, 8, 6, 8. May, 3, 8, 3, 3, 7, 3, 6, 8, 8, 8, 8, 8. June, 7, 7, 7, 7, 2, 7, 3, 3, 3, 3, 8, 3, 6, 3, 3. July, 6, 6, ?, ?. Aug., 3, 4, 7, 1, 7, 7, 1, 5, 5, 6, 3. Sept., 6, 5, 8. Oct., 9. Nov., ?. Dec., 4, ?, ?, 7, 2.	Feb., 8. Aug., 7, 6.	Oct., 28, 28. Nov., 8, 7, 28.	
1852.	Jan, R.S., R.S. Feb., R.S., R.S., R.S., R.S., R.S., R.S., R.S. Mar., R.S., R.S. April, R.S. May, R.S.	Mar., R.S., R.S., R.S., R.S., R.S., R.S. Apr., R.S., R.S., R.S., R.S., R.S., R.S., R.S. May, 27. June, 27, 27. July, R.S., R.S Oct., 27. Nov., R.S.	Feb., 11, 17, 17. March, 6, 6, 6, 17, 6. May, 12. July, 6.	Jan., 14. Feb., 9. Mar., 8, 1, 9, 5. April, 14, 9, 14, 9. May, 9. July, 14.	April, 3, 6. May, 6. June, 7, 3. July, 6, 7.	March, 3, 3, 7. April, 7, 7.	April, 8, 8, 8.	March, ?. May, 24, 9, 10. June, 28.
1853.	—	Jan., R.S., R.S. Feb., R.S., 5, ?. March, ?. April, R.S. May, R.S., R.S., R.S. June, 19, R.S. July, 19, R.S. Aug., R.S.	April, 2.	March, ?. July, 1.		Dec., 6.		

Dr. Kilgour mentions one instance in which typhus was followed by relapsing fever.[i] With these exceptions, in most of the recorded cases, the attack of relapsing fever has preceded that of typhus. Of 45 cases collected by Wardell, in 40, the relapsing fever was followed by typhus; eight individuals in one family had relapsing fever, and in six it was followed by typhus.[k] Cormack observed, that convalescents from relapsing fever were frequently seized with typhus, and that he had noted 19 cases where patients went through unequivocal attacks of both fevers; but he did not say that in any of the cases the attack of typhus came first.[l] Jackson speaks of typhus following relapsing fever at Leith, but does not allude to relapsing fever following typhus.[m] Douglas mentions four instances of patients attacked by both relapsing fever and typhus during their stay in hospital, but does not state which attack came first.'[n] Dr. Jenner says that 'the subjoined cases' illustrate the facts that typhus does not protect from relapsing fever, and relapsing does not protect from typhus; but, probably owing to some omission, in all the three instances given, the relapsing fever preceded the typhus.[o] Steele, in his statistical account of the Glasgow Infirmary for 1847, in which year as many as 2,333 cases of relapsing fever, and 2,399 of typhus were admitted, observes, 'An important character observ-'able from the records, was the immunity which individuals 'enjoyed from relapsing fever, who had suffered previously from 'an attack of typhus. Cases of typhus following in the track of 'relapsing fever, were by no means uncommon, while there is not 'one instance recorded of the former epidemic being followed by 'the latter.'[p] Dr. Hudson, of Navan, in 1847, reported 12 cases where relapsing fever was followed by typhus, but none where the order was reversed.[q] Dr. R. Paterson, in his account of the same epidemic at Edinburgh, observes that many of the patients, after passing through relapsing fever, took typhus; but he nowhere alludes to convalescents from typhus contracting relapsing fever.[r] Dr. W. Robertson, however, stated: 'In a few 'instances, convalescents from relapsing fever became affected 'with typhus, while convalescents from typhus contracted relap-'sing fever, before being dismissed from the wards.[s]

The evidence adduced, shows that while typhus has not unfrequently followed upon relapsing fever, cases in which the order of

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[i] KILGOUR, 1844, p. 323. [k] WARDELL, 1846. [l] CORMACK, 1849.
[m] R. JACKSON, 1844, p. 421. [n] DOUGLAS, 1845. [o] JENNER, 1850, xxiii. p. 119.
[p] STEELE, 1848. [q] *Irish Report*, 1848, viii. 67.
[r] R. PATERSON, 1848, pp. 392, 399. [s] ROBERTSON, 1848, p. 569.

events has been reversed, have been comparatively rare; now the cases where the relapsing fever has come first, do not absolutely prove the non-identity of the two fevers. Contrary to the statement of Henderson, it is now well-known, that one attack of relapsing fever confers no immunity from a second attack (page 312); and, therefore, on the supposition that typhus is only a severe form of relapsing fever, it would not be extraordinary that an attack of relapsing fever should confer no immunity from typhus.

With regard to some of the cases, where the attack of typhus has come first, it may be doubted if the first attack was not enteric fever. It was probably so in one instance referred to by Alison, where there was 'threatening of ulceration of the bowels;' and it is worth mentioning that I have found no notice of typhus followed by relapsing fever at Glasgow, where the distinctions between typhus and pythogenic fever were recognized before 1843, whereas, several are said to have occurred at Edinburgh, where, until lately, these distinctions have received less attention. This explanation, however, does not apply to all the instances above referred to. Wardell gives the details of one case, in which relapsing fever came on a fortnight after what appears to have been an unquestionable attack of typhus; and probably several of the other cases were equally conclusive. Now, although one attack of typhus may not confer an absolute immunity from subsequent attacks, it rarely happens that an individual has two attacks in the same epidemic, and there are few instances on record, where one attack has followed another, with such rapidity as in the cases in question.[t] It must, therefore, be admitted, that an attack of typhus does not confer so great an immunity from an attack of relapsing fever, as from a subsequent attack of typhus, and if this inference be correct, it constitutes a strong argument in favour of the non-identity of the two fevers. On the other hand, there are some grounds for believing that an attack of typhus protects the system more from relapsing fever, than relapsing fever protects from typhus—a supposition rendered more probable by the fact, that in 1843, when Henderson's and Wardell's observations were made, the chances of contracting relapsing fever far exceeded those of contracting typhus.

I am inclined to think, that if the views already advocated, as to the relative etiology of relapsing fever and typhus, be correct, they afford some explanation of the circumstances in question. Relapsing fever being the result of destitution alone, and typhus the result of over-crowding and destitution combined, consequently an attack of typhus protects more from a subsequent attack of

[t] Case xvi. p. 181, is the only exception with which I am acquainted.

relapsing fever, than relapsing fever protects from itself or from typhus. It is also possible, that the protecting effect of an attack of typhus from subsequent attacks of relapsing fever, may be greater when the patient has been suffering from starvation before the attacks of typhus. In the only two cases, where I can find any observation on the point, the patients had not been in want.

That grave objections may be raised to the opinion that a contagious fever can be generated by mere destitution, is readily conceded. It may be well then, to refer to the phenomena known to be exhibited by the living body in consequence of starvation. The effects of starvation on birds and mammals have been studied by Chossat,[u] and on the human subject, by Holland,[v] Donovan,[x] and others.[y] Chossat found that animals rapidly diminished in weight, while, at the same time, the temperature of their bodies decreased. The fat was almost completely removed, and the blood was reduced to one-fourth of its normal amount; whilst the nervous system experienced scarcely any loss. Death appeared to be coincident with the consumption of all the disposable combustible material, and to be really caused by cold; in some cases, it was preceded by cerebral symptoms, showing that ultimately, the nutrition of the nervous centres became impaired.

In Chossat's experiments, the reduction of food was more sudden and complete than it usually is in the human subject. Holland, who investigated the effects of starvation on the poor of Manchester, mentions, among the earliest symptoms, emaciation, exhaustion, languor, listlessness, despondency, and giddiness. These symptoms were sometimes succeeded by others of a cerebral character, such as staggering, dimness of sight, delirium, stupor, and coma. At other times, the exhaustion was followed by symptoms of reaction—quick pulse, flushing of the face, dry tongue, intolerance of light, pains in different parts of the body of a neuralgic character, and delirium. At the same time he observed, that all the secretions of the body became vitiated. Similar effects were witnessed by Dr. Donovan, among the Irish peasantry in the district of Skibbereen, during the famine of 1846-7. In addition, he says: 'The skin exhaled a peculiar and offensive fetor, and was 'covered with a brownish filthy-looking coating, almost as inde- 'lible as varnish; this I was at first inclined to regard as incrusted 'filth, but further experience has convinced me, that it is a secretion 'poured out from the exhalants on the surface of the body.' Other observers have noticed, that during starvation the body exhales a

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[u] Chossat, 1843. [v] Holland, 1839. [x] Donovan, 1848.
[y] Carpenter's *Principles of Human Physiology*, 5th ed. p. 57.

putrid odour, not unlike that of a corpse, and that after death putrefaction is immediate and rapid.[z] Under prolonged abstinence then, the human body seems to become the subject of purely chemical changes, the processes of vital renewal not taking place as in health;[a] febrile symptoms are developed; while, at the same time, the deficient supply of new histogenetic materials, appears to check the elimination of those which have become effete, for in no other way can we account for that tendency to putrescence, manifested during life in the fetid exhalation and peculiar secretion from the skin, and after death in the rapidity with which putrefaction supervenes. It seems not altogether unreasonable to infer, that starvation of itself can generate a febrile state of the system, which is communicable from the sick to the healthy by means of the vitiated exhalations from the body. Mr. Kelly, in his report of relapsing fever at Mullingar, in 1847, wrote as follows: 'Its smell was peculiar, not fetid or heavy, but somewhat 'like burning straw, with a musty odour; and, strange to say, 'there was not a single pauper in the workhouse, with whom 'I had any intercourse, that did not evolve *a similar odour* when 'heated, even by the slightest exertion.'[b]

It may be argued, that persons are constantly exposed to want, without fever resulting. But, under ordinary circumstances, the means provided for the relief of the poor, prevent that degree of want necessary to give rise to the phenomena above described, which are only produced during seasons of famine or of public calamity, when the ordinary means of relief are inadequate. Even then, the effects may often be warded-off by extraordinary exertions on the part of the rich, as was the case for a time in the Edinburgh epidemic of 1843 (see page 315), and, as it is hoped, will be the case with the present 'cotton famine' in our manufacturing districts. Again, there may be other circumstances conducive to, or necessary for, the production of relapsing fever from destitution. In most of the accounts of Irish epidemics of relapsing fever, and in that of Silesia, it is stated that the inhabitants were not only starving, but that they subsisted on unwholesome articles of diet, such as the roots of trees, grass, fungi, etc. (see pages 38, 313, 316). Or, it is possible, that personal uncleanliness may contribute towards the production of the results in question. Relapsing fever has been for the most part confined to the lower Irish, and to the poor of those nations who most resemble the Irish in their habits.

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[z] CARPENTER, *Op. cit.* p. 57.
[a] See LIEBIG's *Letters on Chemistry*, Eng. ed. 1851, p. 323.
[b] *Irish Report*, 1848, viii. 65.

Personal squalor, however, will not alone generate relapsing fever; for, while the former is constant, the latter only appears during seasons of distress.

With regard to the view that there is some subtle and obscure atmospheric agency, which is the cause of both famine and fever, it has been shown that artificial famine has sometimes been followed by the same consequences as that from failure of the crops, and it is unintelligible that any atmospheric agency should only attack the destitute, and leave those who are well-fed almost entirely exempt.

Of all the causes that can be assigned for the origin of relapsing fever, it seems to me that destitution is the most tenable. 'We 'give the name,' says Brown, 'of cause to the object, which we 'believe to be the invariable antecedent of a particular change;'[c] and such appears to me to be the relation of destitution to relapsing fever. But whether or not they stand in the relation to one another of cause and effect, the intimate connection between them is indisputable. The facts bearing on this point are most important, even if the theory founded on them be not accepted.

Section VI.—Symptoms of Relapsing Fever.

A.—Clinical Description.

The patients, while walking about, or engaged in their ordinary avocations, or on first awaking in the morning, without any premonitory symptoms, are suddenly seized with a sense of chilliness and with rigors, oftentimes severe, and accompanied by frontal headache, and pains in the back and limbs. There is slight prostration of strength from the first; but it rarely approaches in severity to that of typhus; the patients usually take to bed at once, owing to extreme giddiness, rather than weakness; very often they are able to walk to hospital two or three days after their seizure.

After a period, varying from a quarter of an hour to several hours, the cold stage is succeeded by a dry burning skin, great increase of the headache and of the pain in the back and limbs, and violent thirst. Occasionally, on the second or third day, there is sweating, in some cases profuse and lasting for several hours, but not attended or followed by any relief to the headache, and other symptoms. In a few cases, this sweating occurs earlier: no well-marked hot stage intervens between it and the primary rigors, but the sweat breaks out on the face and upper part of

[c] *Inquiry into the Relation of Cause and Effect*, 3rd ed. Edin. 1818.

the body, while the patient is yet in his initiatory rigors. In many cases, the sweating alluded to is not observed.

After the primary cold stage, or after the above-mentioned sweating, when it occurs, the skin continues dry and hot (102 to 107° Fahr.), this condition being occasionally interrupted by irregular short rigors, or slight sweating. No characteristic eruption appears at any time; but in a large proportion of the cases, there is decided jaundice, and in others, the skin exhibits a bronzed hue. The pulse almost invariably exceeds 100; as a rule, it reaches 120; and, in not a few cases, it is as high as 140 or 160; it is not rarely 140 on the second day of the disease; at the same time it is often full, and of considerable firmness. The tongue is at first moist, and covered with a white or yellowish fur; it may continue in this state throughout the illness; but, in many cases, after three or four days, it becomes dry all over, or exhibits a dry brownish streak along the centre. The thirst is excessive; the appetite is often absent; at other times it is voracious; the bowels are constipated. In the jaundiced cases, there is no absence of bile from the stools, which either retain their normal hue, or are unusually dark. In many cases, there is more or less tenderness on pressure over the epigastrium, and in the splenic and hepatic regions; while percussion indicates obvious enlargement of both liver and spleen. Nausea and vomiting are not uncommon. Sometimes they are amongst the earliest symptoms, and continue incessant. The vomited matters consist of a green bilious fluid, or occasionally are black, like coffee-grounds. The urine is high-coloured, and, in the jaundiced cases, contains bile. The headache continues severe, and is often of a throbbing character, while the pains in the muscles and joints are often intense. Sleeplessness is almost invariably a most distressing symptom; the mind is usually clear, but in a few cases, violent delirium occurs about the fifth or sixth day.

About the end of the first week, sometimes as early as the third, or as late as the tenth, but, in most cases, on the fifth or seventh day, there is an abrupt cessation of all the symptoms. At one moment, the patient may be groaning with pain, with his pulse at 120 or 150, and a dry burning skin, and within a few hours, the pulse may have fallen to below 70, and the temperature from 107° to 98° Fahr., the skin is moist, and the tongue clean, and the patient, free from pain, declares himself perfectly well, with the exception of a certain amount of languor and exhaustion. This sudden amelioration is almost invariably ushered in by profuse perspiration (in some instances with an eruption of miliary vesicles),

but, in rarer cases, by diarrhœa, epistaxis, catamenial discharge, or hæmorrhage from the bowels. Occasionally, improvement is immediately preceded by brief, but violent, delirium.

The patient has now a good appetite; and, day by day, he gains strength, and there are all the indications of permanent convalescence, except that the pulse is often unusually slow—45 to 60. In many cases he is up and walking about, or he is discharged from hospital. But, after a week's interval, mostly on the seventh day from the crisis, or on the fourteenth (twelfth to the twentieth) day from the commencement of the first attack, without any warning or cause to account for it, what is called *the relapse* sets in. The patient is suddenly seized with rigors, followed by headache, pains in the back and limbs, burning skin, very rapid pulse, furred tongue, vomiting, tenderness at the epigastrium, constipation, and occasionally delirium. The rise of the pulse with the relapse is as rapid as was its fall in the preceding crisis. In a few hours it may rise from 50 or 60, to 120, or upwards. The relapse is, in fact, a repetition of all the symptoms observed in the primary paroxysms: sometimes the symptoms are more severe; at other times, they are less so. The relapse usually lasts three days; in some cases, it lasts only one or two days, and in others, five or more.

Occasionally, there is a second relapse, coming on about the twenty-first day, and lasting two or three days; and, in rare instances, even a third or fourth relapse occurs. In other cases again, there is no relapse at all, the patient continuing to convalesce after the crisis of the first attack.

Relapsing fever is far from being mortal. Uncomplicated cases almost invariably recover; and the total mortality rarely exceeds 1 in 25, or 1 in 50. Great prostration and sinking, however, are apt to come on suddenly in the course of some cases;—the face assumes a purplish hue; the extremities are cold and livid; the patient cannot be roused, and there are all the phenomena of profound collapse, which may terminate in death: sometimes a fatal termination occurs in this way, within a few hours after there had been no evidence of danger. At other times, death occurs at the end of the first or second paroxysm, from suppression of urine, with delirium, coma, and occasionally convulsions. Pregnant females, invariably abort in the course of relapsing fever, sometimes in the first, but oftener in the second paroxysm. Abortion is sometimes, but not invariably, a cause of death.

After the termination of the paroxysm, convalescence is often retarded by the occurrence of dysentery, severe muscular and arthritic pains, or ophthalmia.

B.—Analysis of Principal Symptoms.

a. The Physiognomy.

The countenance is often flushed; but the flushing is red or purplish, and rarely of that dingy, earthy hue so common in typhus, and not circumscribed as in enteric fever. The vascularity and suffusion of the eyes, are also less marked than in typhus. The expression, towards the close of the paroxysm, sometimes betokens languor and depression. The stupid, confused expression, so common in typhus, is rarely met with in relapsing fever; but in the few cases, where cerebral symptoms supervene, the countenance may assume all the characters of the typhoid state, common to many diseases.

The presence of jaundice in a considerable number of cases, imparts a peculiarity to the countenance, not observed in other fevers of temperate climates.

Dr. Cormack described as one of the most remarkable peculiarities of the epidemic of 1843, 'a bronzing, leadening, or purpling 'of the countenance, before and after seizure.' In the ordinary mild cases, the countenance of the patient, according to him, had a peculiar appearance, which might be designated '*bronzed*,' for want of a better term; whereas, in the severe cases, 'a deep, per-'sistent purple colour of the face appeared before or immediately 'after the invasion of the disease'[d] These phenomena were chiefly observed at the commencement of the epidemic. After the epidemic had reached its climax, Cormack stated, that facial bronzing ceased to be met with. Other observers of the epidemic failed to recognize these appearances.[e]

When Relapsing Fever proves fatal by sudden sinking, death is often preceded by a deep dusky hue of the face, and a deep purple colour of the nose. When the purple nose is combined with jaundice, the patient presents a truly frightful aspect.[f]

b. Morbid Phenomena referrible to the Skin.

1. *Eruption.*—Relapsing Fever in Great Britain and Ireland, is not characterized by any eruption. Neither the measly eruption of typhus, nor the lenticular rose spots of pythogenic fever, are ever present. The existence of the latter has never been asserted; and I can find mention of only 4 cases in which an eruption like that of typhus has been said to have been present, while the negative-evidence is overwhelming. Cormack, although he maintained, that

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[d] Cormack, 1843, pp. 3, 23. [e] See, for example, Douglas, 1845, p. 209, and Wardell, 1846. [f] Graves, 1848, i. 286.

the typhus eruption 'was absent in almost every case,' recorded one instance where he believed Relapsing Fever had presented a typhus rash, and came to the conclusion, 'that there is such a thing 'as persons being occasionally affected with the measly eruption, 'in addition to the usual symptoms of Relapsing Fever.'[g] This patient, however, had an attack of 'genuine and unequivocal 'typhus' a few weeks after,[h] and Dr. Cormack, in an appendix to his work, admitted that he had been mistaken in the case, and declared that the eruption of typhus was never present in Relapsing Fever.[i] Dr. Arrott, of Dundee, met with one instance where an 'eruption resembling measles' was present;[k] a third case, where 'the apparently true typhoid measly eruption appeared, and lasted 'for three or four days,' occurred in the practice of Dr. Watson, of Leith;'[l] and a fourth case was reported by Dr. W. Robertson, of Edinburgh.[m] It is possible, as suggested by Wardell in reference to Arrott's case, that the eruption in these instances was urticaria, a well-known accompaniment of jaundice, or roseola; for Douglas observed one case of Relapsing Fever, where, on the sixth day, the body became covered with a bright rose-coloured eruption, disappearing on pressure, and 'different from that of typhus;'[n] and, in none of the instances quoted, are the spots said to have run the usual course of those of typhus. On the other hand, Alison,[o] Henderson,[p] and Craigie,[q] all testified to the universal absence of typhus eruption in the Relapsing Fever at Edinburgh, in 1843; while Wardell examined upwards of 1200 cases,[r] and Douglas 220, without ever detecting it. Jackson failed to find it once in upwards of 800 cases which came under his notice at Leith;[s] and with the exception above referred to, Arrott never saw it in 672 cases observed at Dundee. Jenner never found any eruption in the cases examined by him in London, between 1847 and 1850,[t] and there has been no eruption in any of the cases which have come under my own notice.

But although Relapsing Fever has not been characterized by any eruption in Great Britain or Ireland, Dümmler, Virchow, and other German writers, all agree in stating, that a copious eruption was far from uncommon in the Relapsing Fever of Silesia in 1841. It differed from that of typhus, in the following particulars: it

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[g] Cormack, 1843, pp. 73, 106. [h] Wardell, 1846, xxxvii. 953.
[i] Cormack, 1849. [k] Arrott, 1843, p. 129.
[l] Jackson, 1844, p. 430. [m] Robertson, 1844. In Robertson's case of 'Betsy Gibb,' the 'relapse' was possibly due to pleurisy. [n] Douglas, 1845, p. 218.
[o] Alison, 1843. [p] Henderson, 1843. [q] Craigie, 1843.
[r] Wardell, 1846. [s] Jackson, 1844, p. 430. [t] Jenner, 1850, xxii. 647.

appeared as early as the second or third day, and after one or two days, disappeared; it was rosy or pale red, effaceable by pressure, followed by desquamation, and not obvious after death.[u] It is possible, that the eruptions observed in exceptional cases by Douglas, Cormack, and others in this country, was of a similar nature, and that its early appearance and evanescent character, may have prevented its more frequent detection in this country. This point deserves investigation in future epidemics.

2. *General Hyperæmia.* Lividity of the dependent parts of the body is much rarer than in typhus. But, in cases where there are pulmonary complications, much cerebral oppression, or sudden sinking, a purple mottling of the whole surface, and considerable lividity of the face may be observed (see *Physiognomy*).

3. *Petechiæ, Purpura-Spots, and Vibices.* True petechiæ (see page 129), varying in size from a pin's head to a split pea, but in most cases very minute, are not uncommon. Smith noted them in 314 out of 1000 cases at Glasgow.[x] In many instances, these minute petechiæ are evidently flea-bites. Alison was of opinion that even the larger spots 'originated in flea-bites, and extended 'by little ecchymoses.' They cannot always, however, be thus accounted for. They often make their first appearance in large numbers in one night after the patient's admission into hospital; their size is occasionally much larger than flea-bites; while Wardell, Henderson, and Smith examined them carefully with a lens in a number of instances, but could not discover a central punctum. Jackson also caused two patients, suffering from the fever, (a severe attack in both instances) to be bitten by a number of fleas confined in a bottle. The bites went through the ordinary stages of a flea-bite in a healthy person, and did not enlarge. There can be little doubt then that these petechiæ are often the result of a hæmorrhagic tendency, engendered by the fever, or by the previous anæmic condition of the patients. Paterson met with petechiæ chiefly in persons who had been in the greatest destitution.[y] They differ from the petechiæ of typhus in not being developed in the centre of exanthematous spots. They do not appear on any specific day, but they are more common in the first paroxysm than in the relapse, and in cases where there is jaundice than when jaundice is absent; of 21 petechial cases observed by Jackson, 14 had jaundice.[z] Occasionally, they co-exist with hæmorrhages from the mucous surfaces; and Alison mentions

[u] See VIRCHOW, DÜMMLER (p. 349), &c., 1849; also *Review*, 1851, p. 35.
[x] SMITH (2), 1844, p. 70.
[y] R. PATERSON, 1848, p. 404.
[z] JACKSON, 1844, p. 428.

one instance where the serum in a blister-vesicle was perfectly black.[a]

Vibices are occasionally observed, and then the case is usually severe; but the minute petechiæ are probably not of much importance in prognosis. Although Kilgour, Alison, and Jackson thought that they were more frequent in fatal than in mild cases, they are far from uncommon in the mildest cases, while Douglas at Edinburgh,[b] and Smith at Glasgow,[c] were both of opinion that they added in no way to the danger or severity of the disease.

4. *Sudamina.* An eruption of miliary vesicles occasionally accompanies perspiration at the period of crisis. Ormerod found this eruption so common in London in 1847, that he designated the disease 'Miliary Fever.'[d] Few other observers, however, have noted their occurrence, and they were present in only 12 of 220 cases examined by Douglas.[e]

5. *Herpes.* (See *Complications*).

6. *Desquamation.* An attack of relapsing fever is sometimes followed by extensive desquamation. My friend, Dr. Gueneau de Mussy tells me, that he once removed from the body of a young lad, convalescent from relapsing fever at Dublin, a piece of epidermis fully ten inches square.

7. *The Temperature* of the skin is, as a rule, higher than in typhus. In the epidemic of 1817-19, according to Christison, it ranged from 102° to 105°, and at times it even reached 107.[f] Careful observations on the temperature in relapsing fever are, however, still a desideratum. The skin often communicates to the hand a hot, pungent feel (*calor mordax*), and the patient's perception of a dry, burning heat is sometimes intense.

8. *Moisture.* One of the most characteristic features of relapsing fever is the profuse perspirations which, in most cases, usher in and accompany the crises. The patients for some hours are literally bathed in perspiration. Slighter perspirations are occasionally observed in the course of the paroxysm, as for instance, on the second or third day, or immediately after the primary rigors. The perspiration which accompanies the crisis is sometimes preceded by a slight rigor, and in rare instances by a slight fall of the pulse. As a rule, however, the pulse does not fall until the sweating begins. The perspiration is described by Cormack as having 'a characteristic disagreeable smell,' and an acid reaction.[g]

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[a] Alison, 1843. [b] Douglas, 1845, p.217. [c] Smith, 1844 (2), p.70.
[d] Ormerod, 1848, p. 217. [e] Douglas, 1845, p.218.
[f] Christison, 1858, p. 583. [g] Cormack, 1843, p.4.

9. *Odour from the Skin.* (See page 325).

c. Morbid Phenomena presented by the Organs of Circulation.

1. *The pulse* in most cases exceeds 100. During the first two days, it may vary from 90 to 112, but after this, and sometimes before, it reaches 120 or upwards, while in not a few cases as the disease advances, it is 140 or even 160, and in rare instances 170 or 180. Of 220 cases examined by Douglas in 1843, the pulse exceeded 120 in 105. In 20 of these 105 cases, the pulse exceeded 140 in the minute; in 29 it was above 130, but under 140; and, in 56, above 120, but under 130.[h] This remarkable rapidity of the pulse, although most common in children, is likewise observed in adults. Of the 20 patients observed by Douglas, in whom the pulse exceeded 140, several were above 40 years of age. Again, the pulse may attain this great rapidity on the second or third day of the disease, and, in this respect, as was shown by Henderson, relapsing fever presents a marked contrast to typhus. In 15 cases of typhus, observed on or before the fifth day, Henderson found the average frequency of the pulse to be exactly 100, whereas of 38 cases of relapsing fever the average frequency of the pulse during the first five days was 123: in the 15 cases of typhus, the pulse exceeded 104 in only two instances; in the 38 cases of relapsing fever, it did so in 37.[i] Moreover, the high pulse does not in itself indicate danger. Of the 220 cases noted by Douglas 19 died; but only one-third of the deaths occurred among patients in whom the pulse exceeded 120, and not one among those in whom it exceeded 140. Of 9 cases observed by Henderson, where the pulse exceeded 135, only one died. On the supposition, that relapsing fever is but a mild variety of typhus, it is not a little remarkable, that a symptom which, in typhus, is thought to indicate danger, is so common in relapsing fever where the mortality is so small.

The rapidity with which the pulse falls at the period of crisis is also remarkable. In a few hours, it may fall from 140 to 60. During the interval between the two paroxysms, the pulse is in many cases, singularly low, often not exceeding 45 or 50, and from this, with the supervention of the relapse, it may rise to 120 or upwards, in an equally short space of time, to that in which it had previously fallen. In the cases where the pulse is remarkably slow, assuming the erect posture will sometimes raise it from 50

[h] DOUGLAS, 1845, p.213. [i] HENDERSON, 1843, p.206.

to upwards of 100. The slow pulse is not due to any slowness in the contraction of the heart, but to a prolongation of the pause.

During the febrile paroxysms, the pulse is often full and bounding, and it is usually of better strength than in typhus. It is rarely intermittent or irregular, except towards the close of fatal cases. After the cessation of the paroxysms, the pulse is always weak.

2. *Action of the Heart.* The diminished impulse and impairment, or absence of the first sound, indicative of softening of the left ventricle, so common in typhus, are not observed in relapsing fever. Stokes mentions one case where there was absence of the first sound, which was probably due to temporary weakness, and not to softening, as the sound speedily returned under the use of stimulants.[i]

Drs. Stokes,[k] Lyons,[l] and Heslop,[m] however, have drawn attention to the frequent occurrence of a systolic bellows-murmur in cases of relapsing fever, which is rarely, if ever, produced by true typhus.[n] This murmur was usually loudest at the base of the heart, and along the great vessels, and always diminished in intensity, or became altogether imperceptible, when the patient sat up or assumed the erect posture. In some cases, it was heard in both paroxysms, and remained during convalescence; but it always disappeared, as the patient regained strength. From these characters, it was obviously a blood-murmur, independent of valvular disease, and, as such, it is interesting in reference to the connection shown to exist between starvation and relapsing fever. Occasionally, when there is no distinct murmur, the first sound is prolonged to almost double its normal length.

3. *Blood.* (See *Post-Mortem Appearances*).

d. Morbid Phenomena, presented by the Organs of Respiration.

1. *The Respiratory movements* are usually quickened to an extent corresponding to the acceleration of the pulse, so that occasionally they amount to 40 in the minute, independently of any pulmonary complication. When, however, the pulse falls to below the normal standard (e.g. 40 or 50), there is no corresponding fall of the

[i] *Diseases of the Heart*, 1854, p. 427.
[k] Ibid. p. 423.
[l] Lyons, 1861, pp. 105, 161.
[m] Ibid.
[n] Stokes records two cases of maculated fever accompanied by systolic murmur, but says that such cases are very rare. One of the cases was probably an example of Enteric Fever (*Op. cit.* p. 432).

respiratory movements: so that the ratio of the respirations to the pulse may be as 1 to 2.

In cases, where there is great pain in the epigastric or hypochondriac regions, with enlargement of the liver and spleen, the respirations may be mostly thoracic, or sometimes interrupted.

2. *Cough and Expectoration.* (See *Pulmonary Complications.*)

3. *The Expired Air* in Relapsing Fever has not been examined.

e. Morbid Phenomena presented by the Organs of Digestion.

1. *The Tongue* is usually from the first covered with a white, yellowish, or brownish fur, of varying thickness; but a clear triangular space is sometimes observed at the tip, which, as well as the edges, is occasionally redder than natural. In mild cases, the fur is often a mere film, or the tongue remains natural throughout the attack; in rare instances, it is red and glazed, though moist. In the majority of cases, it continues moist throughout the attack; but, in others, about the third or fourth day, it presents a dry brownish streak along the centre; or it becomes dry all over, or, in rare instances, dry, brown, and thickly crusted. The latter appearance is only seen in very severe or fatal cases.

Of 200 cases, in which the appearances of the tongue were noted by Douglas, it was moist throughout the attack in 119; in 74 of the 119, it was merely covered with a slight film, or was almost clean; while in 45 there was a whitish, yellowish, or brown fur along the centre, or diffused over the whole surface. In 81 cases the tongue became dry: in 28 of these, the dryness was limited to a central streak: the whole surface was dry, but without crust, in 30: while in 23, the tongue was dry and brown, and in some of the 23, thickly crusted. Of the cases in which the tongue was partially dry, 7 per cent. died; and of the cases where the tongue was dry all over, with or without crust, death occurred in 16 per cent.[o]

2. *Brown Sordes* occasionally collect on the teeth and lips in severe cases, when the tongue is dry and brown; but they are much rarer than in typhus.

3. *The Appetite* usually ceases with the supervention of the paroxysm, returns during the intermission, and ceases again during the relapse. Loss of appetite, however, is not invariable. Not uncommonly the appetite has been observed to be voracious during the paroxysms, as well as in the interval. The following

[o] Douglas, 1845, p. 214.

extract from the Report of the London Fever Hospital for 1843, has reference to the Relapsing Fever of that year:—

'A peculiarity, very novel in its character, was an inordinate 'desire for food; this desire, so very unusual in fever, was all but 'universal. In some instances, it was so uncontrollable, that no 'representation of the danger of indulgence produced the slightest 'effect in pacifying the minds of the patients; but many insisted 'on leaving the hospital long before their convalescence was suffi- 'ciently advanced, declaring that they preferred running all risks 'to enduring their constant sense of starvation. Several of these 'were brought back to the hospital in a few days, having relapsed 'into a hopeless state of fever.' In Glasgow, Dr. Perry stated that a day or two before the occurrence of the relapse, the patients often exhibited a great increase of appetite, or craving for food.[p] Observations to the same effect were made in Ireland in 1847. Thus, Dr. Russell, of Enniskerry, in his description of relapsing fever, remarked: 'One of the most anomalous symptoms of the 'epidemic, one which marked its true character (a famine fever), 'and impressed on the mind of the attendant the source and origin 'of the disease, was the importunate calls for food by all pauper 'patients, even during the first days of the attack.[q]

4. *Thirst* is an almost invariable symptom, and is excessive and insatiable in a far larger proportion of cases than in typhus.

5. *Nausea and Vomiting* are very common symptoms. Vomiting occurred in 643 of 1000 cases observed by Smith, at Glasgow.[r] It is often one of the earliest symptoms, as in 56 of 80 cases observed by Wardell,[s] and then it may subside after two or three days, or recur more or less frequently. Sometimes, it is incessant throughout the whole paroxysm, everything swallowed being immediately rejected. Occasionally, it does not appear until the paroxysm has lasted for several days. It always ceases with the crisis, and may or may not return with the relapse. In some cases, it is more severe in the relapse than in the first paroxysm.

The vomited matters consist for the most part of green bile, or of the ingesta, tinged green of various shades. 'Black vomit,' similar to what occurs in 'Yellow Fever,' has been described by several observers, but more particularly by Cormack,[t] Arrott,[u] and Wardell.[x] It is not noted as having been observed in any epidemic except that of 1843; and then it occurred only in a few cases,

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[p] Perry, 1844, p. 82. [q] *Irish Report*, 1848, viii. 64. [r] Smith (2) 1844, p. 69.
[s] Wardell, 1846. [t] Cormack, 1843. [u] Arrott, 1843. [x] Wardell, 1846.

although it seems to have varied in frequency at different places. Cormack and Wardell met with several unequivocal examples of 'black vomit,' at Edinburgh; but Alison[y] and Douglas,[z] who had extensive opportunities of watching the epidemic in the same city, did not meet with a single case. Craigie,[a] writing at the very height of the epidemic (October, 1843), stated, that up to that time only 2 or 3 cases altogether had been observed in Edinburgh; and Wardell himself remarked, that the cases in which this symptom occurred were quite exceptional. Dr. Smith, of Glasgow, seemed to doubt if true black vomit ever occurred; while Perry of Glasgow, and Kilgour of Aberdeen, make no mention of it. On the other hand, Dr. Arrott described this symptom as 'quite common' in the fever at Dundee. Arrott gives no detailed description of his cases; but the cases observed by Cormack and Wardell appear to have been unequivocal examples of true black vomit, the appearance being due to blood extravasated from the capillaries of the stomach, and altered by the acid secretions. In some cases, there was a fine inky sediment in the vomit: at other times the sediment was grumous, in consistence like thick hare-soup, and in colour varying from dark-brown to black. Moreover, the opinion that these appearances were due to altered blood, was confirmed by the sources of the extravasation being found after death, in the form of superficial ecchymoses, and large clots of blood in the submucous areolar tissue of the stomach and intestines. Dr. W. Reid, of Glasgow, records the case of a girl, aged 14, who vomited large quantities of clotted blood. In this case, there was also hæmorrhage from the bowels and from the ears.[b]

Both Cormack and Wardell looked upon 'black vomit' as an almost fatal sign. The former only observed it in the most 'malignant' cases; and all the few cases seen by the latter, died. Of 16 fatal cases in the Dundee Infirmary, black vomit was noted in 6. At the same time, if true black vomit was so common in Dundee, as stated by Arrott, it is remarkable that the mortality from the disease in that town was even less than at other places. Arrott lost only 7 of 672 patients, and in 1 only of his fatal cases does there seem to have been black vomit.

Of the 6 fatal cases of black vomit occurring in the Dundee Infirmary, it is worth noticing that the patients were mostly advanced in life; the youngest was 25; the oldest, 69; and the average age of the 6 was 44 years, or considerably above the average age at which relapsing fever usually occurs (see page 303).

[y] Alison, 1844 (1). [z] Douglas, 1845. [a] Craigie, 1843.
[b] Reid, 1843, p. 359.

6. *Meteorism* is an occasional symptom, and is apt, when accompanied by enlargement of the liver and spleen, to be the source of no small distress.

7. *Gurgling*, on pressure, may be felt in cases complicated with diarrhœa; but it is, on the whole, rare, and is not confined to any particular part of the abdomen.

8. *Abdominal Pain and Tenderness.* In almost all cases, there is more or less pain, increased by pressure, in the epigastric and hypochondriac regions. The pain, in many cases, is slight, but in others, it is so acute as to cause great uneasiness, and interfere with respiration. Frequently, it is confined to the epigastrium; but, at other times, it is limited to either hypochondrium, or it may extend over all these regions. Severe lancinating pains in the left side are not unfrequently found associated with enlargement of the spleen. Pain and tenderness in the epigastrium often accompany vomiting, but their severity is not necessarily proportionate to the urgency of the vomiting. Epigastric pain, associated with vomiting, was present in 273 of 450 cases observed by Wardell, and the proportion would have been greater, had all the cases been observed from the commencement. There is no tenderness on pressure over the iliac regions, except where dysentery exists as a complication.

9. *Enlargement of the Liver and Spleen.* Pains in the left hypochondrium are often complained of, when it is impossible to detect any enlargement of the spleen, and are then perhaps muscular. But not uncommonly, palpation and percussion furnish distinct evidence of great enlargement of the spleen, and such enlargement may exist without pain or tenderness. In some instances, the organ is so large, that its edge can be felt projecting several inches beyond the lower margin of the left ribs, or it may cause a visible bulging of the abdominal wall. This enlargement may occur during the paroxysms, or after they have ceased; and in the latter case, it has been noticed to be ushered in and accompanied by a symptomatic fever of its own, differing from a paroxysm of the primary fever, in [that it yields speedily to the local remedies directed against the spleen (cupping, etc.,) and in subsiding as the swelling disappears. When the enlargement occurs during a paroxysm, it does not necessarily subside with the crisis, but may continue throughout the intermission, though it often disappears spontaneously and rapidly after the crisis. It has been a subject of discussion, whether the enlargement of the spleen in relapsing fever be due to acute inflammation or simple hyperæmia. The fact, that it sometimes occurs after the cessation of the paroxysm,

and is then accompanied by symptomatic fever, which subsides under treatment directed against the spleen, has been thought to point to inflammation; but too little is known of the anatomical characters of acute splenitis as distinguished from hyperæmia, to enable us to come to a decision, even were opportunities of inspecting the organ, under the circumstances in question, more common.

Enlargement of the liver also occurs, but is less common and extensive than that of the spleen. It appears to be due to simple hyperæmia.

10. *Constipation.* As in typhus, the bowels are, as a rule, constipated, and are with difficulty moved, although, diarrhœa coming on late in the disease is an occasional complication, or seems to have a critical character.

11. *Characters of the Stools.* The stools may retain their natural colour and consistence; more commonly, they are darker than natural. In the most severe cases, black coffee-ground matter similar to that which is occasionally vomited, or black stools are passed *per anum.* Dr. Gibson, of Glasgow, met with 9 instances (out of 202 cases), where hæmorrhage took place from the bowels.[c]

12. *Jaundice* is a symptom noticed by almost all writers on relapsing fever, but is not so frequent as might be inferred from the importance attached to it by some observers. It was observed by Welsh in the Edinburgh epidemic of 1817-19. 'Decided yellow-'ness of the skin and eyes,' he remarks, 'occurred in 24 of 743 'cases (or in 1 in $30\frac{9}{2}\frac{3}{4}$); and in all those cases where the experi-'ment was tried, the urine tinged linen yellow.'[d] This estimate was probably under the mark, as regards relapsing fever, as it included a few cases of typhus. Jaundice was also noticed in the epidemic of 1826;[e] but, although there are no data for ascertaining its precise frequency, it does not seem to have been more common than in the epidemic of 1843. Many observers of the latter epidemic furnish precise information on the point. Thus, jaundice was present, according to

Wardell,[f] (Edinburgh)	in	78	of	955	cases,	or in	1	of 12·24.
Douglas,[g] ,,	,,	29	,,	220	,,	,,	1	,, 7·58.
Jackson,[h] (Leith)	,,	31	,,	300	,,	,,	1	,, 9·7.
Gibson,[i] (Glasgow)	,,	13	,,	114	,,	,,	1	,, 8·77.
D. Smith,[k] ,,	,,	384	,,	1000	,,	,,	1	,, 2·6.
Total . . .		535	of	2589			1	of 4·84.

[c] Gibson, 1843, p. 332. [d] Welsh, 1819, p. 73. [e] Graves and Stokes, 1826.
[f] Wardell, 1846. 34 cases of typhus have been deducted from Wardell's calculation. [g] Douglas, 1845. [h] Jackson, 1844. [i] Gibson, 1843.
[k] Smith, 1844, p. 69.

It is possible, that Wardell's estimate, as regards Edinburgh, was too small, in consequence of his observations not commencing until the epidemic was at its height. His own tables show that there was a progressive diminution of the yellow cases, as the epidemic advanced. Thus, of 320 cases in the Edinburgh Infirmary, in October, 1843, when the epidemic was at its height, 37, or 1 in 8·65 were yellow, but of 426 cases admitted in January, 1844, 28, or 1 in 15, were yellow, and of 80 patients in April, when the epidemic had nearly ceased, only 2 had jaundice. In the early part of the epidemic, jaundice was probably more common, although Henderson,[l] and Craigie,[m] speak of the symptom as being even then exceptional. At other places, jaundice appears to have been more frequent than at Edinburgh. At Glasgow, according to Dr. D. Smith, it occurred in 2 out of every 5 patients, and at Dundee, it is also said to have been more common than at Edinburgh.

In 1847-8, Robertson[n] says that at Edinburgh, jaundice was less common than in 1843, and Paterson noticed it only in 4 of 141 cases;[o] but at the same time, in London, Jenner met with it in nearly one-fourth of his cases.[p]

It would appear then, that although jaundice varies greatly in frequency at different times and places, it rarely occurs oftener than once in 4 cases, and occasionally it is much more uncommon.

It is met with both in the old and in the young, but is most common at the middle period of life.

The jaundice rarely appears before the third, fourth, or fifth day of the primary paroxysm. It may occur during the first paroxysm only, or in the relapse only, or in rare cases it is seen in both paroxysms, and does not disappear in the interval. It may commence at the height of the pyrexia, or at the time of crisis. Of 28 cases, observed by Douglas,[q] the jaundice occurred in the first paroxysm only, in 16; 2 of the 16 patients became jaundiced on the fourth day; and none earlier than this. In 10 cases, the jaundice only occurred in the relapse; and in 2 cases it was present in both paroxysms. Jackson found jaundice in the first attack only, in 13 cases; in the second only, in 18 cases; in the third only, in 2 cases; and in both the first and second attacks, in 2 cases.[r] As a rule it does not last more than a few days.

The intensity of the jaundice varies from a slight tinge to a

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[l] Henderson, 1843. [m] Craigie, 1843. [n] Robertson, 1848, p. 373.
[o] R. Paterson, 1848. [p] Jenner, 1850, xxii. 646. [q] Douglas, 1845, p. 216.
[r] Jackson, 1844, p. 426.

deep yellow. Of 29 cases, Douglas noted it as intensely bright in 11, complete but less intense in 9, and very faint in 9.[8]

The conjunctivæ are first tinged, and then the neck, face, chest, arms, abdomen and lower extremities. In the jaundiced cases, the serum in the vesicle raised by a blister contains bile, and the urine is occasionally found so loaded with it, as to resemble porter. There is no impediment, however, to the flow of bile into the intestine, for the fæces retain their natural colour, or are unusually dark, and, moreover, the bile-ducts after death are found to be pervious. Dr. Alison stated, on the authority of Dr. Peacock, that, in some instances, the bile was thick and viscid so as apparently to cause obstruction, but this condition is far from constant, and in many instances the bile is perfectly fluid, and is found in the duodenum in abundance.

Most observers have agreed in making jaundice a formidable symptom in relapsing fever. In the epidemic of 1817-19, Welsh observed jaundice in 4 out of 34 (1 in $8\frac{1}{2}$) fatal cases, but only in 20 of 709 (1 in 35) cases which recovered. In 1826-27, jaundice was looked upon by Graves and Stokes as a very fatal symptom. In 1843, Cormack regarded it as characterizing the most malignant cases; 4 out of 8 jaundiced cases under Craigie died; Alison observed jaundice in most of the cases which proved fatal under his care; and this symptom was present in all the 16 cases which were fatal during the epidemic in the Dundee Infirmary. Among the symptoms which accompany the jaundice, vomiting, and more or less pain in the epigastric and hypochondriac regions, especially the right hypochondrium, are the most common, while in the more severe cases, 'black vomit,' delirium, coma, subsultus, and other cerebral symptoms are occasionally met with. Delirium was noticed by Douglas in 6 out of 29 jaundiced cases (1 in 5) but only in 12 of 191 (1 in 16) non-jaundiced cases.

On the other hand, jaundice is met with in a large number of instances, which differ in no other circumstance from the most mild cases, a number in fact far larger than that in which it is attended by dangerous symptoms. Welsh spoke of jaundice as 'a very trifling occurrence;' of 6 cases that came under Henderson's notice, 1 patient died from a totally different complication, and the other 5, in all of whom the jaundice was well marked, 'had not a single symptom that made them differ 'from the ordinary cases, excepting the yellowness.' According to Douglas, 'vomiting was not more frequent or troublesome in

[8] Douglas, 1845, p. 216.

'the cases with jaundice than in the ordinary cases;' of 35 cases of jaundice under Jackson, only 2 died; while at Dundee, where jaundice was said to have been more frequent than elsewhere, the total mortality was very much less, or only 1 in 96. Alison remarked, that 'many jaundiced cases had the crisis at the usual 'time, and went on quite favourably with little treatment.' Moreover, jaundice is far from being a constant accompaniment of delirium and other cerebral symptoms. Of 18 cases in which Douglas observed delirium, only 6 were jaundiced.

It follows that, although jaundice has been observed in a large proportion of severe and fatal cases of relapsing fever, it is not in itself a dangerous symptom, and it is probably unconnected with the dangerous symptoms by which it is occasionally accompanied. Since the days of Galen, it has been the custom to look on the bile as possessed of narcotic properties, and as capable of producing coma, delirium, and other cerebral symptoms, when absorbed into the blood; and, even at the present day, this opinion is commonly entertained. But it is well known, that, in jaundice, from obstruction of the ducts, the above-mentioned symptoms are rare, while the experiments of Frerichs show, that the artificial introduction of bile into the blood is not followed by the symptoms usually attributed to it, and that its presence in the blood is harmless.[t] Moreover, cerebral symptoms and death are common in other fevers where jaundice is rarely observed, whereas, in relapsing fever, where jaundice is so common, cerebral symptoms are comparatively uncommon, and the mortality is peculiarly small. Indeed, the observations of Henderson[u] and Mr. Michael Taylor,[x] render it very probable, that, in relapsing fever, as in typhus, it is to urea (and other products of tissue-metamorphosis usually excreted by the kidneys), and not to bile, that the dangerous cerebral symptoms, which occasionally supervene, must be attributed.[y] In the only fatal case complicated with jaundice that occurred under Dr. Henderson's care, death was preceded by cerebral symptoms, and urea, in considerable quantity, was found in the serum of the blood. From observations on other cases, Dr. Henderson was inclined to believe that cerebral symptoms, in relapsing fever, were always due to a similar cause.

The jaundice occasionally observed in relapsing fever, is merely one of the results of the absorption of a poison into the blood.

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[t] *Klinik der Leberkrankheiten*, Syd. Soc. Transl. i. 187, 395.

[u] Henderson, 1843. [x] Taylor, 1844.

[y] Even in true yellow fever, the cerebral symptoms admit of a similar explanation. (See page 173.)

The poisons of other fevers, such as those of yellow fever, remittent and intermittent fevers, typhus and pythogenic fevers, act in like manner, although some give rise to jaundice oftener than others. Other poisons, with the nature of which we are better acquainted, produce similar effects, such as the poison of pyæmia, and that of certain snakes. How these poisons act it is difficult to say; but it is not improbable, as suggested by Frerichs, that they interfere with the normal metamorphoses which ought to take place in the blood, and cause the biliary acids naturally present in that fluid to be transformed into bile-pigment, instead of being converted into urinary pigment.

f. Morbid Phenomena presented by the Urinary System.

Relapsing fever has scarcely been met with since the introduction of the volumetric means of analysing urine. Researches are, therefore, still wanting on the characters presented by this fluid; but, as far as we know them, they are full of interest.

The *quantity* of urine, during the paroxysms, varies with the amount of fluid ingesta; but, as a rule, is equal to, or exceeds, the normal standard. After the termination of the paroxysms, the quantity is usually much increased; in a considerable number of cases, Dr. Henderson found it to vary from 48 to 80 ounces in the 24 hours.[z]

At the same time, during the paroxysms, the *colour* is dark, and the *specific gravity* usually high, and it may, therefore, be inferred as was suggested by Henderson, that the quantity of urea and uric acid is much increased.[a] The total amount of *urea* excreted in twenty-four hours has not yet been ascertained; but, if the theory be true, that the amount of urea formed in the system depends on the degree of febrile action, the quantity is greater in relapsing fever than in typhus. That the quantity is abnormally large, is shown by an observation of Dr. Henderson's, where urea was found in the serum of the blood, while, at the same time, the urine, which amounted to about twenty fluid ounces, yielded abundant crystals of nitrate of urea when treated with nitric acid, without having been previously evaporated. Even in Bright's disease, urea can rarely be detected in the blood, unless the urine excreted has for some time been reduced to below one-third, and, at the same time, its proportion of urea been greatly diminished. It is, therefore, probable, that there was an increased formation of urea in the case in question.[b]

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[z] HENDERSON, 1843, p. 224. [a] Ibid. p. 224; see also WARDELL, 1846, xxxix. p. 547; also PARKES, *On the Urine*, 1860, p. 260.

[b] HENDERSON, 1843, p. 224.

But occasionally the urine presents remarkable deviations from the characters now described. Its quantity is reduced, or it is altogether suppressed, while the amount of urea is more or less diminished. This change may occur towards the close of the paroxysms, in the interval, or after the termination of both, although oftenest at the stage of crisis; but whenever it occurs, it is found to be accompanied by cerebral symptoms, such as delirium, stupor, coma, or convulsions. In fact, while it admits of demonstration, that cerebral symptoms occurring in relapsing fever, are independent of cerebral inflammations, or of obvious organic lesion within the cranium, there are grounds for believing that they are due to the retention in the blood of urea and other products of tissue-metamorphosis. A brief recapitulation of the evidence in favour of this view may not be out of place.

1. Henderson mentions the case of a gentleman who was seized on the day of crisis with uneasy sensations in the head, and confusion of mind, and for eighteen hours passed no urine. Ten grains of nitre were prescribed every hour. He began immediately to pass abundance of urine, and the symptoms were at once relieved.[c]

2. In a second case, under Dr. Henderson, the commencement of cerebral symptoms was accompanied by suppression of urine, and death was preceded by several attacks of convulsions. Urea was obtained by Dr. Douglas Maclagan in considerable quantity from the blood, and in smaller quantity from the serum of the cerebral ventricles.[d]

3. In a third case, also under Dr. Henderson, the commencement of cerebral symptoms was marked by a reduction of the urine to one-half its former amount. Mr. Michael Taylor ascertained that the total urea excreted in the urine did not exceed 109·3 grains,[e] (the normal average, according to the lowest estimate, being 286 grains); and Dr. Maclagan obtained urea from blood taken from the arm.[f]

4. In a case under Dr. Wardell, the occurrence of cerebral symptoms was accompanied by suppression of urine, and abundance of urea was found in the blood by Mr. M. Taylor.[g]

5. In a fifth case of relapsing fever, with cerebral symptoms, recorded by Mr. Taylor, the urine did not exceed 16 ounces, and urea was discovered in considerable quantity in the blood.[h]

6. In another case observed by Mr. Taylor, the development of

[c] Henderson, 1843, p. 222.
[d] Ibid.
[e] Taylor, 1844, p. 293.
[f] Henderson, 1843, p. 223.
[g] Wardell, 1846, xxxix. 547.
[h] Taylor, 1844, p. 293.

cerebral symptoms was accompanied by a reduction of the urine to 16 ounces, the total amount of urea in twenty-four hours not exceeding 174 grains.[i]

7. Cases of suppression or diminution of urine, with cerebral symptoms, were observed by Jackson at Leith;[k] and by other writers on the epidemic of 1843.[l] Suppression of urine was also noted as a very fatal symptom of relapsing fever in 1847.[m]

Copious deposits of *lithates*, about the period of crisis, are more common than in either typhus or enteric fever.

The urine of relapsing fever has not often been examined for *albumen*; but, as far as we know, this substance is rarely present, even when urea is found in considerable quantity in the blood. In the third case quoted from Henderson, the urine was free from albumen; while, in the second, it did not coagulate with nitric acid, and the kidneys, after death, appeared moderately hyperæmic, but healthy.

In the jaundiced cases, bile-pigment is found in greater or less quantity; but *leucine* and *tyrosine* have yet to be sought for.

The condition of the *chlorides*, *sulphates*, and *phosphates*, is unknown.

g. Morbid Phenomena presented by the Nervous and Muscular Systems.

1. *Head-ache*, to a greater or less degree, is invariably complained of, and is usually one of the first symptoms. The pain is mostly in the forehead. In some cases it is slight, and continues only for a day or two; but, as a rule, it is severe, and lasts throughout the paroxysm, subsiding with the crisis, but returning with the relapse. The head-ache is much more severe, and oftener of a shooting, darting, or throbbing character, than that of typhus.

2. *Vertigo.* Most patients suffer from great giddiness, as well as head-ache, from the very commencement of the attack (see pages 326 and 349).

3. *Muscular and Arthritic Pains.* A remarkable and most distressing symptom of relapsing fever, is the severe pains in the muscles and joints, complained of by most patients. They occur in many cases during the paroxysms: but are most common and severe in the interval between the paroxysms and during convalescence, when the patient in other respects is in perfect health. They were very common in the relapsing fever of 1817—19,[n]

[i] Taylor, 1844, p. 293.
[l] See Wardell, 1845, case vii. &c.
[n] Welsh, 1819, p. 18.
[k] Jackson, 1844, pp. 423, 431.
[m] *Irish Report*, 1848, viii. 300.

and in that of 1826,[o] and are mentioned by almost every writer on the epidemic of 1843. They were said to be more common at the commencement of the epidemic of 1843, than subsequently. Wardell found that 438 of 536 patients, or upwards of 4 in 5, suffered from these pains.[p] They are sometimes seated in the muscles of the trunk or extremities; at other times in the larger joints, or in the feet. During convalescence, they may take the form of sharp stitches in the sides. In character, they are not unlike the pains of acute rheumatism, and are often most excruciating. They are not attended, except in rare instances hereafter referred to, by any swelling or redness of the joints. Their cause is obscure, but they possibly depend on the presence in the blood of some abnormal substance, such as uric or lactic acid.

Relapsing fever is a disease attended altogether by more pain and general uneasiness than typhus, which are also more impressed on the memory, from the circumstance that the mind is usually clear, and the perception unimpaired. Persons who have passed through both fevers, invariably look back on the former as the source of greatest distress.

4. *Impairment of the Mental Faculties.—Delirium.* In relapsing fever, delirium is an exceptional symptom; and, in most cases, the mind remains clear throughout the attack. Occasionally, the patient talks a good deal in his sleep, and has frightful dreams, but he is easily roused, and gives rational answers. Of 220 cases observed by Douglas, delirium occurred in only 18, or about 8 per cent: of these 18 cases, in 6 the patients had previously been intemperate; and in 1 case it was apparently due to opium.[q] When delirium does occur, it is oftener acute and noisy than in typhus.

About the period of crisis, or after the termination of the paroxysm, the patients sometimes become stupid and confused, with a tendency to stupor; at other times, they become suddenly and violently delirious, and cannot be kept in bed. These symptoms may persist, and gradually merge into those of the 'typhoid 'state' (dry brown tongue, muttering delirium, and more or less unconsciousness), or they may speedily pass off. The connection between these cerebral symptoms, and diminished excretion by the kidneys, has been already referred to. The delirium which occurs at the period of crisis, is sometimes remarkable for its sudden outbreak, its violent character, and its very short duration.[r] Dr. Robertson mentions an instance, where the patient had conversed

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[o] O'Brien, 1828, p. 530. [p] Wardell, 1846, xl. p. 107.
[q] Douglas, 1845, p. 211. [r] Jackson, 1844, p. 420; Robertson, 1848.

with him rationally at the time of his visit, but scarcely had Dr. R. left the ward, when he became suddenly outrageous, screamed, raved, abused his attendants, could with difficulty be restrained in bed, and passed his stools and urine involuntarily. Within fifteen minutes he was again calm and collected, bathed in perspiration, and in perfect oblivion as to what had just passed. Dr. Robertson met with 5 or 6 instances of this nature at Edinburgh in 1847-8, and at Dublin they were said to be more common.[s]

5. *Wakefulness, Somnolence, Coma*, etc. Sleeplessness is a very common and distressing symptom, both in the paroxysms and in convalescence, in the latter case being usually due to the severity of the muscular and arthritic pains.

Stupor and coma, so common in typhus, are rare in relapsing fever. Their occasional appearance, in connection with suppression of urine, has been already referred to. Under such circumstances, they usually supervene at, or after, the period of crisis. When they come on before the cessation of the paroxysm, and do not speedily pass off, there is no well-marked crisis, and all the phenomena of the 'typhoid state' may be gradually developed.

6. *Prostration.* More or less prostration is present in all cases from the first, but in most cases it is slight in comparison to that of typhus, and it is rarely so complete as to prevent the patient getting out of bed, or helping himself, except in those instances where syncope or cerebral symptoms supervene. It is vertigo, rather than muscular prostration, that causes patients to take to bed at an early stage of the disease.

9. *General Convulsions* are occasionally observed in those cases where other cerebral symptoms supervene, at or after the period of crisis. They have also been known to occur unexpectedly in cases which seemed to be progressing favourably, at or after the crisis, independently of other head-symptoms. The cases where they occur are usually fatal. Of 4 cases alluded to by Henderson, 2 died; and the result in the other 2 is not stated.[t] Jackson records the case of a boy, who recovered, after having had two 'convulsive fits' on the day of crisis, in which, for twenty minutes, 'the limbs became rigid, the body motionless, and the eyes turned 'upwards.' The pathology of convulsions, in relapsing fever, is probably the same, as in typhus (see page 162). In the only case where attention has been directed to the matter, urea was found in considerable quantity in the blood and in the fluid of the cere-

[s] Robertson, 1848, p. 373. [t] Henderson, 1843, p. 221.

bral ventricles. The urine was not coagulable by nitric acid. After death, the kidneys were found to be 'of ordinary size and 'consistence, moderately loaded with blood, and, when washed, 'seemingly a little paler than usual in some places.[u]

7. *Muscular Paralysis.* Retention of urine, and the involuntary passage of urine and fæces, are rare, except in cases characterized by sudden syncope or by cerebral symptoms. Involuntary evacuations were noted by Douglas in only 6 of 220 cases, and, in several, the discharges were due to extreme diarrhœa, rather than to paralysis; all 6 died.[x]

8. *Tremors, Subsultus, Carphology,* and *Rigidity* of the muscles are also rare symptoms. Tremors mostly occur in persons of dissipated habits; the other symptoms are only observed in those rare instances where the disease passes into the typhoid state.

h. Morbid Phenomena referrible to the Organs of Special Sense.

1. *Organs of Vision.* The 'ferretty eye,' or the injected condition of the conjunctivæ, so characteristic of typhus, is comparatively rare in relapsing fever. The pupils are, for the most, natural; but, in cases where stupor and other cerebral symptoms supervene, they are often contracted.

2. *Organs of Hearing.* Deafness is not a common symptom. In 220 cases, Douglas met with it only 12 times; and in 8 cases it was very slight, and only lasted a day or two. Of the 4 cases, where it was decided, it occurred early in the attack in one, and in the remaining 3, it only came on in convalescence.[y]

3. *Organ of Smell.* Epistaxis is not uncommon, and is occasionally profuse. It may occur at any stage of the paroxysms. Sometimes it is one of the earliest symptoms; but it is most common at the period of crisis, when it now and then appears to take the place of the ordinary perspirations. Douglas noted epistaxis in 13 of 220 cases at Edinburgh, and many of the other patients stated that they had bleeding from the nose before admission.[z] In many of the Irish epidemics, epistaxis has been very common. (See page 171).

4. *Cutaneous Sensibility.* Hyperæsthesia is not met with in relapsing fever (see page 171); but, occasionally, the jaundiced patients complain of itchiness, which is a well-known accompaniment of jaundice, under all circumstances.

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[u] Henderson, 1843, p.222.
[x] Douglas, 1845, p.210.
[y] Douglas, 1845, p.210.
[z] Ibid. p.220.

Sect. VII. Stages and Duration of Relapsing Fever.

UNLIKE typhus, relapsing fever is divisible into well-marked stages. In ordinary cases, there are four:—The primary paroxysm, the intermission, the relapse, and convalescence. The paroxysms are again subdivisible into the accession, the pyrexial stage, and the crisis.

1.—*The Mode of Accession.*

The mode of accession is, in most cases, sudden, without any premonitory symptoms. The patient, on awaking in the morning, or when sitting at the fireside, or walking, or engaged in his ordinary avocations, is suddenly seized with a sense of chilliness and with rigors, which are much more severe than those sometimes observed at the commencement of typhus. These rigors are often accompanied by a sensation of cold trickling down the back, frontal headache, severe pains in the back and limbs, nausea or vomiting. From Wardell's observations on the epidemic of 1843, it would appear that in 103 out of 120 cases, the invasion was marked by distinct rigors; in 31 out of 40 cases, by headache; in 56 out of 80, by nausea or vomiting; and in 52 out of 80, by arthritic or muscular pains.[a] In some few cases, sickness is the first symptom, and this, with headache, pains in the back and chilliness, precedes the attack of rigors for two or three hours. In rare cases, there are no well-marked rigors, but only a sense of chilliness.

Premonitory symptoms are far from frequent; they were noted by Douglas in 5 only out of 220 cases, although it is admitted that in some of the cases they may have been overlooked. These symptoms were anorexia, general pains, and a feeling of debility and *malaise*.[b]

As a result of the suddenness of the invasion, patients not unfrequently apply for admission into hospital on the first or second day of the disease. Of 80 cases observed by Wardell, the average duration of the fever before admission was 4·7 days. At the same time, the prostration is not so great as to prevent many from going about for two or three days, and when patients take to bed on the first day, it is oftener due to giddiness than to weakness.

2.—*Duration of the Primary Paroxysm.*

The duration of relapsing fever has been spoken of by all observers as short, when compared with that of typhus; and hence,

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[a] Wardell, 1846. [b] Douglas, 1845, p. 11.

the designations, 'Short Fever,' 'Five Days' Fever,' and 'Seven Days' Fever,' have been given to it. These designations, however, apply only to the first paroxysm, and do not include the relapse, which occurs so frequently as to justify its being regarded as part of the disease.

As to the primary paroxysm, in the epidemic of 1739-41, Rutty[c] fixed its ordinary duration at five, six, or seven days; Welsh[d] and Christison[e] assigned five days as the usual limit to the fever of 1817-19; and O'Brien five or seven days to that of 1826.[f] In the epidemic of 1843, Cormack made five days the ordinary limit;[g] but most other observers thought seven days the more common duration,[h] and Jackson, at Leith, found that the crisis occurred in most cases on the eighth day.[i] In the epidemic of 1847, the common duration at Edinburgh, according to Paterson, was five days,[k] and according to Robertson, seven.[l] Elaborate statistics bearing on this point are given by the authorities referred to. It suffices here to state, that the most common duration of the primary paroxysm is from five to seven days; that in rare imstances it does not exceed three or four days, and that probably in no case, except where complications exist, does it exceed ten days. Douglas's observations seem to show, that the average duration is less below thirty years, than at a more advanced period of life, and less in females than in males.

Craigie,[m] Cormack,[n] Smith,[o] and other writers on the epidemic of 1843 allude to a slight remission on the third day of the first paroxysm, consisting in a slight abatement of the headache and thirst, with slight perspiration, but rarely with any fall in the pulse. This remission is not constant, and not a characteristic feature of the disease. Douglas failed to observe it.

3. *The Intermission.*

After the cessation of the primary paroxysm, the patient usually expresses himself as in perfect health, and in uncomplicated cases, with the exception of debility, an abnormally slow pulse, or muscular and arthritic pains, he is free from all complaint. Day by day, he recovers strength, and, by the end of a week, he may be up and going about, or may have resumed his work. He often feels

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[c] Rutty, 1770, pp. 75, 90. [d] Welsh, 1819, p. 78. [e] Christison, 1858, p. 582.
[f] O'Brien, 1828, p. 527. [g] Cormack, 1843, pp. 5, 100.
[h] Alison, 1843, p. 1; Douglas, 1845, p. 12; Wardell, 1846, xxxviii. pp. 155, 196; Kilgour, 1844, p. 322. [i] Jackson, 1844, p. 421.
[k] R. Paterson, 1848, pp. 391-5. [l] Robertson, 1848, p. 373.
[m] Craigie, 1843, p. 416. [n] Cormack, 1843, p. 5. [o] Smith, 1844, p. 70.

so well, that it is difficult to persuade him that he has not yet shaken off his malady. Dr. Christison relates an amusing anecdote in reference to his colleague, Dr. Bennett, who was attacked with relapsing fever on the first outbreak of the epidemic of 1843, when the disease was unknown except to the older members of the profession. Dr. Christison saw him after the termination of the first paroxysm: 'Though still confined in a great measure to bed 'from debility, he was well otherwise, and enjoying the genuine 'pleasures of a fever convalescent. When he had detailed to me 'his case, I told him he had sustained, to all appearance, an 'attack of my old acquaintance synocha (relapsing fever), whose 'face I had not seen for a good many years; that he was not yet 'done with it, and that he would have another three days' attack, 'commencing with rigor on the fourteenth day. Dr. Bennett, 'surprised—I will not say incredulous—replied, that the relapse 'had no time to lose, as there were only three or four hours of the 'fourteenth day to run. It did, indeed, lose no time, for I must 'have scarcely reached home from his house, before the rigor 'set in with violence; and he had three days of fever again, 'terminating, as the primary attack had done, with an abrupt 'crisis by sweating.' [p]

The ordinary course of events, then, is, that, after an interval of a week from the crisis of the first paroxysm, all the febrile symptoms return. In many cases, the interval is exactly seven days, so that the relapse occurs on the twelfth or fourteenth day, according to the duration of the primary paroxysm, and can be predicted with tolerable certainty. Douglas ascertained the average duration of the intermission in 59 cases, to be 7·15 days.[q] At the same time, its duration is not invariable; it may not exceed two or three days, while, in a few cases, it extends to ten or twelve days, or even longer. According to Douglas, the relapse does not occur sometimes until after the 21st day from the primary seizure.[r] Lyons states, that, in the cases of relapsing fever observed in the Crimea, the period of intermission was remarkably inconstant, varying from two to many days.[s] According to O'Brien[t] and Douglas,[u] the cases where the primary paroxysm is longest, have also the longest intermission; and, as a rule, Douglas found the intermission longer in males than in females.

Occasionally, the intermission of febrile symptoms is not quite complete, or there is an intermission rather than a remission.

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[p] Christison, 1858, p. 591. [q] Douglas, 1845, p. 15.
[r] Douglas, 1845, p. 19. [s] Lyons, 1861, p. 107. [t] O'Brien, 1828, p. 528.
[u] Douglas, 1845, p. 15.

The pulse does not fall to its normal standard, the appetite does not return, and the patient complains of lassitude, slight headache, and giddiness, and has occasional chills and perspirations. Such cases, however, are exceptional (in only 15 of 220 cases observed by Douglas); and in most, if not all, there is probably some local complication. Again, in those cases where cerebral symptoms supervene at the period of crisis, the intermission may be masked, and the fever may have a more protracted character; but even then the crisis is often marked by sweating and a considerable fall in the pulse and temperature.

Lastly, in some cases, permanent convalescence follows the crisis of the first paroxysm, and there is no relapse.

4. *Relapses.*

On or about the fourteenth day from the primary seizure, subject to the variations already mentioned, the patient is a second time attacked with rigors, followed by a repetition of the symptoms which characterized the first paroxysm. The second attack, like the first, comes on suddenly, and without warning. Kilgour remarks that, at Aberdeen, it was preceded by loss of appetite and sleeplessness;[x] but Perry tells us that, in Glasgow, he found the appetite before the relapse unusually acute;[y] and, in most instances, there are certainly no premonitory symptoms of any sort.

The second attack may be milder or more severe than the first. Cases are met with, where the first attack is mild, and the second is characterized by delirium, diarrhœa, dysentery, or other grave symptoms. But more commonly the second attack resembles the first, or runs a milder course. Occasionally it is indicated by nothing more than a slight increase of the pulse and temperature.

The duration of a relapse varies from a few hours to several days; the average is usually from three to five days, or less than that of the primary paroxysm. In some cases, the relapse lasts less than twenty-four hours; and, in a few, it is prolonged to seven or eight days; but it is rarely longer than this in uncomplicated cases. Of 112 cases investigated by Douglas, the relapse lasted less than twenty-four hours in 3, one day in 18, two days in 25, three days in 34, four days in 25, five days in 4, six days in 2, and seven days in 1.[z] In the Crimea, according to Dr. Lyons, the relapse was occasionally protracted to twenty-one days.[a]

As stated already, a relapse is not invariable. Of 182 cases under Dr. Craigie,[b] relapses occurred in 110. Of 300 cases under

[x] Kilgour, 1844, p. 322. [y] Perry, 1844, p. 82.
[z] Douglas, 1845, p. 19. [a] Lyons, 1861, p. 107. [b] Craigie, 1843.

Jackson, of Leith, 3 died during the first attack, and, of the remainder, all, save 21, relapsed.[c] Of 1,000 cases under Dr. D. Smith, at Glasgow, 712 relapsed;[d] and of 946 cases observed by Wardell, at Edinburgh, 603 had one or more relapses.[e] Adding these results together, it follows that, of 2,425 cases, relapses occurred in 1,701, or in upwards of seven-tenths. Several observers of the epidemic of 1843 remarked, that the relapses became less frequent towards its close. Thus Wardell found that in October, 1843, 72 out of 80, had relapses, but in April, 1844, only 40 out of 80.[f] Steele also observed, that, towards the termination of the epidemic of 1847, at Glasgow, relapses became less frequent, until, at last, they formed the exception rather than the rule.[g] Relapses, however, are probably much more common than might be inferred from the above data. Some patients are only admitted into hospital in the relapse; a still larger number are dismissed before the relapse occurs; while, in others, the relapse is so mild, that it is apt to be overlooked. Douglas and Cormack were disposed to to think that, in 1843, few or no cases escaped without relapsing.[h]

Occasionally, a second relapse, lasting three or four days, occurs from the twenty-first to the twenty-fourth day (counting from the primary seizure), sometimes, however, as early as the eighteenth, or as late as the thirtieth day. A second relapse was observed, by Wardell, in 67 of 946 cases; by Jackson, in 28 out of 297; and by Douglas, in 11 out of 220,—altogether, in 106 out of 1,463 cases, or in 1 out of 14. The second relapse commences and terminates in the same way as the two preceding paroxysms. The symptoms are almost invariably mild in their character. The attack may last from one to ten days, but rarely exceeds forty-eight hours.

A third, fourth, and even a fifth relapse have occasionally been observed, making in all six paroxysms. In the above 1,463 cases, a third relapse occurred nine times, or in 1 out of 162 cases, and a fourth relapse once. These relapses usually resemble a common febricula.

From what has been stated, it follows, that, under ordinary circumstances, when there are but two paroxysms, the total duration of relapsing fever amounts to nearly three weeks.

5. *Crisis.*

The paroxysms of relapsing fever usually terminate by a well-

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[c] Jackson, 1844, p. 421. [d] Smith (1), 1844, p. 72. [e] Wardell, 1846, xxxix, 274. [f] Ib. [g] Steele, 1848. [h] Douglas, 1845, p. 15; Cormack, 1843, p. 87.

marked crisis, which, in the majority of cases, is characterized by copious perspiration. In many instances, the sweating is preceded by chilliness, or a slight rigor. It lasts for some hours, and is followed by sudden and marked relief to all the symptoms, the pulse falling, perhaps, from 140 to 70, and the temperature from 106° to 98° Fahr. Other discharges, such as diarrhœa and dysentery, epistaxis, copious menstruation, or, in rare instances, hæmorrhage from the bowels, may occur at the same time; but it is seldom that they entirely displace the sweating. The crisis is sometimes characterized by considerable languor and prostration, or by the cerebral phenomena already alluded to (page 346).

6. *Convalescence.*

Although relapsing fever is a much less formidable disease than typhus, convalescence is usually much slower. Many patients remain for a long time very weak, and complete recovery is more apt to be retarded by the occurrence of distressing sequelæ, than in typhus.

Section VIII.—Complications and Sequelæ of Relapsing Fever.

a. Diseases of the Respiratory Organs.

The morbid conditions of the respiratory organs are the same as in typhus, but are less severe, and seldom interfere much with recovery.

1. *Bronchitis* is not uncommon, but is usually slight, except when of old standing. Smith noted it in 132 out of 1000 cases at Glasgow, in 1843, the cases being most numerous in winter and spring.[i] According to Arrott it was very common in the same epidemic at Dundee.[k]

2. *Hypostatic Consolidation* is comparatively rare; but—

3. *Pneumonia* is more common than in typhus. According to Jenner, it is the next most common complication after enlargement of the liver and spleen.[l] Smith met with 3 cases; Alison with 1 case;[m] and Douglas with 6 out of 220 cases. Of the last 6 cases, 5 died; but in 4, there was also inflammation of the bowels.

4. In rare instances, pneumonia terminates in *gangrene.* One case was observed by Douglas.

5. *Pleurisy* is also an occasional complication.[n]

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[i] Smith, 1844 (1), p. 70. [k] Arrott, 1843, p. 132. [l] Jenner, 1850, xxii. 647.
[m] Alison, 1843, p. 2. [n] Robertson, 1844; Smith, 1844 (1), p. 70.

6. *Laryngitis* was observed by Smith in 9 (of 1000) cases about the period of crisis. It is usually slight, but may require the performance of tracheotomy.[o]

b. Complications referrible to the Organs of Circulation.

1. *Sudden Syncope* comes on in some cases, and may prove rapidly fatal. It may occur in the primary paroxysm, in the intermission, or in the relapse. The pulse becomes small or imperceptible, the whole surface is cold and livid, and the patient is perfectly unconscious of all that takes place. The most extraordinary circumstance is, that these symptoms may come on suddenly, in cases previously mild, and may terminate in death, within a few hours after the patient has been looked upon as in no danger. Douglas mentions three remarkable instances. In one, death occurred a few hours before the first crisis; in a second, death occurred suddenly during the intermission without any previous complaint, the patient being found in the morning, lying in an easy posture, and dead, as if for some hours; the third patient was found dead about the period of the first crisis, without any warning, and within half an hour after having expressed herself as feeling easy.[p] Occasionally, the syncope is due to hæmorrhage, as in two cases recorded by Dr. Cormack,[q] and by Dr. Reid, of Glasgow;[r] but in the majority of cases, no hæmorrhage, or other cause of exhaustion can be discovered. No observation has yet been made in these cases on the impulse and sounds of the heart during life, or on its condition after death. The syncope may possibly be sometimes due to protracted starvation before, and during the fever.

2. *Palpitations* are sometimes complained of during convalescence. They may, or may not, be accompanied by the anæmic cardiac murmur already described.

3. *Hæmorrhages*, from various parts, are by no means uncommon. The most common variety is epistaxis (see page 348). Hæmorrhage from the uterus (page 361), from the stomach (page 337), from the bowels (page 339), and from the ears,[s] may likewise occur. They may appear at any stage of the first paroxysm, or of the relapse, but oftenest at the crisis. Dr. Gibson, of Glasgow, met with hæmorrhages in 21 out of 202 cases: in 8, the bleeding took place from the nostrils; in 1 from the lungs; in 3 from the stomach; and in 9 from the bowels.[t] Dr. Douglas, of

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[o] Paterson, 1848. [p] Douglas, 1845, p. 274. [q] Cormack, 1843, p 41.
[r] Reid, 1843. [s] Reid, 1843, p. 359. [t] Gibson, 1843.

Edinburgh, observed hæmorrhages in 14 out of 220 cases. In 1, it was from the uterus, and in 13, from the nostrils: epistaxis had also occurred in several other cases prior to admission.[u]

c. Complications referrible to the Nervous System.

1. *Partial Palsy*, lasting for a few days or weeks after recovery, is occasionally noticed. Cormack mentions the case of a female, aged 36, in whom loss of power in both deltoids continued for about ten days, after restoration to health in every other respect had taken place.[x] In 2 (of 220) cases, Douglas observed partial paralysis of the fore-arms. In one, it came on during the interval between the attacks. In both cases, the attack was sudden, with accompanying numbness, but with no head-symptoms. The paralysis lasted for several weeks.[y]

2. *Muscular and Arthritic Pains* are more frequent and severe during convalescence, than in the paroxysms. They are, in fact, the most common sequelæ, and often cause great suffering, and prevent sleep; but they usually cease after a few days, when the strength is regained.

d. Complications presented by the Organs of Special Sense.

1. *Post-febrile Ophthalmia.* One of the most remarkable features of relapsing fever, is the frequent occurrence during convalescence of a peculiar disease of the eyes. This sequela has been observed in almost all epidemics, but is never met with after typhus or pythogenic fever. The first cases were described by Mr. T. Hewson, in his work on Venereal Ophthalmia,[a] and occurred in his practice so long ago as 1814. Mr. Wallace,[b] Dr. Jacob,[c] and Dr. Reid,[d] gave an account of the affection as observed at Dublin during the epidemic of 1826. It is alluded to by almost all writers on the epidemic of 1843, and an excellent description of it was published at that time by Dr. Mackenzie, of Glasgow.[e] It was again observed as a sequela of relapsing fever in the epidemic of 1847, at which time also, Dr. Dubois, of New York,[f] described it as occurring among the Irish immigrants recovering from relapsing fever.

The disease presents two distinct stages, the *amaurotic* and the *inflammatory*. During the first stage, there exist amaurotic symp-

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[u] Douglas, 1845, p. 219. [x] Cormack, 1843, p. 148.
[y] Douglas, 1845, p. 272.
[a] *Observations on the History and Treatment of Ophthalmia*, London, 8vo, 1814, pp. 34, 119. [b] Wallace, 1828. [c] Jacob, 1828. [d] Reid, 1828.
[e] Mackenzie, 1843. [f] Dubois, 1848.

toms alone, the patient complains of more or less dizziness of vision, of *muscæ volitantes*, and luminous stars. The length of time that the amaurosis exists before the visible signs of inflammation, is far from being uniform. In some cases, the amaurosis commences with convalescence, or even before the cessation of the febrile paroxysms, and yet the inflammatory stage does not supervene for weeks or months; but still oftener the dulness of vision does not commence for several days, weeks, or even months after the febrile attack, and is then almost immediately followed by the symptoms of inflammation. As a rule, the inflammation commences from three weeks to three months after the cessation of the fever. Occasionally its advent is protracted to four, five, or eight months after the fever, while Douglas mentions two cases where it appeared as early as the second day of the relapse. The inflammatory symptoms are invariably preceded by the amaurotic, and occasionally slight amaurosis is all that is met with, no signs of inflammation manifesting themselves externally. After the inflammation has subsided, the amaurotic symptoms continue for a longer or shorter period. The inflammation appears to commence in the retina, and from this to spread to the iris and sclerotic, the capsule of the lens, the choroid and the lining membrane of the cornea. The conjunctivæ in general are but slightly affected. The inflammation is attended by considerable lachrymation, and by intense pain in and around the eye, aggravated during the night, and preventing sleep. The pulse varies from 84 to 120; rigors are frequent; the tongue is usually clean and moist. Recovery is tedious; in most cases, two months have been necessary to effect a cure, and, unless carefully treated, the disease may terminate in permanent loss of sight.

The disease rarely attacks both eyes, and the right suffers more frequently than the left. Jacob never met with a case in which both eyes were affected. Of Wallace's 40 cases, the right eye alone suffered in 36, the left in 2, and both in 2. Of Mackenzie's 36 cases, the right only was affected in 18, the left in 10, and both together, or consecutively, in 8. Of 29 cases under Dubois, the right only was affected in 15, the left, in 11, and both, in 3. Adding these results together, there are 105 cases, of which the disease was limited to the right eye in 69, to the left in 23, and attacked both in 13.

This ophthalmia has been thought to occur more frequently in females than in males. This was the result of Jacob's experience, and, of Mackenzie's 36 cases, 27 were females. On the other hand, Wallace stated, that males are as often attacked as females, and Dubois had 16 male, to 13 female patients.

It occurs at all ages, but most frequently between 10 and 30. Of Wallace's 40 patients, the youngest was 10, and the oldest 36. Jacob met with no case above 45, and only 3 of 30 cases were above 25. Of Mackenzie's 36 cases, 26 were between 10 and 30. Dubois and Jacob, however, met with cases, aged only $2\frac{1}{2}$ or 3, and Mackenzie mentions others upwards of 50.

There are no data for ascertaining the proportion of cases of relapsing fever which are followed by ophthalmia, as the local disease rarely appears until long after the patient has been discharged from hospital.

Occasionally, the patient seems to have quite recovered from the effects of the febrile attack, before the ophthalmia commences, but far oftener, a considerable degree of debility remains. Jacob and Mackenzie both state, that ophthalmia was most common in the very poor, who had insufficient nourishment during convalescence; and the latter observes, that many of his patients were wan and extremely weak at the time of their attack. These observations point to insufficient nourishment as one of the main causes of the ophthalmia; and if this be so, the circumstance explains why the affection in question succeeds no other fever than relapsing fever. In many instances, exposure to cold seems to be the immediate exciting cause.

2. *Epistaxis* (see page 348).

e. Diseases of the Organs of Digestion.

1. *Pharyngitis.* Welsh[g] states, that, in the epidemic of 1817-19, 'in 181 of 743 cases, the fauces or tonsils were more or less 'inflamed; but in most cases, the affection was slight.'

2. *Diarrhœa and Dysentery* are common complications or sequelæ of relapsing fever, and are among the chief causes of death. They were often observed in the Scotch epidemic of 1843, especially during autumn; in winter and spring they were comparatively rare. Smith met with them in 167 out of 1000 cases at Glasgow,[h] and Douglas in 33 of 220 cases at Edinburgh:[i] putting these results together, they were present in 200 of 1220 cases, or in one-sixth. Most commonly, the diarrhœa comes on in the relapse, or after the cessation of both paroxysms. Of Douglas's 33 cases, looseness came on in the first paroxysm in only 3, and in 2 of the 3 it was very trifling; in 30, it did not commence until after the day of relapse, and one-half of the 30 were not attacked until after the second crisis. Occasionally the diarrhœa appears to have

[g] Welsh, 1819, p. 61. [h] Smith, 1844 (1), p. 70. [i] Douglas, 1845, p. 269.

somewhat of a critical character; in 6 of Douglas's 33 cases, it occurred at the precise time of the crisis, and in 4 of the 6 it lasted only for a single day. At the same time, the diarrhœa does not appear to be substituted for the sweating; in the 6 cases alluded to, sweating was also noted in 4, and, in the other 2, its absence was not positively ascertained; in 2, it was unusually profuse.

These attacks occur at all ages with about equal frequency. Their accession is mostly sudden, and is occasionally preceded by rigors; at other times, it is gradual. They vary in severity; in 11 of Douglas's 33 cases the looseness was trifling, and easily restrained; of the remaining 22 cases, 8 were fatal; in all the fatal cases, the attack did not commence until after the cessation of the relapse. In some cases, there is great pain and tenderness over the lower part of the abdomen, or the patient complains of tenesmus and griping pains. Vomiting, sometimes of an urgent nature, is a common accompaniment. In the milder forms, the stools are fluid, feculent, dark, and very offensive, and rarely contain blood; but in the more severe forms, they are scanty, and consist almost exclusively of blood and mucus. The pulse is seldom quick, except in the paroxysm, and is occasionally remarkably slow and does not exceed 60. The purging may last only a few hours, or several weeks. Douglas mentions one patient who died within seven hours of its commencement, and another whose death occurred on the 25th day of the attack, or the 48th from the accession of the fever.

3. *Peritonitis* is fortunately a rare complication, as it is always fatal. Of 2,846 cases of relapsing fever in the Glasgow Infirmary in 1847-8, 7 died from peritonitis.[k] Paterson mentions a case where death resulted from peritonitis on the sixth day,[l] and Douglas another, where it was fatal on the 38th day. In the last case, the peritoneal surfaces of the bowels adhered at all their points of contact, leaving circumscribed interspaces filled with purulent fluid.[m]

f. Complications referrible to the Integuments and Joints.

1. *Erysipelas* is an occasional sequela of relapsing fever, and is sometimes fatal.

2. *Œdema of the Lower Extremities* is not an uncommon sequela, and appears to depend on debility of the organs of circulation, or on an impoverished state of the blood. It is chiefly met with in

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[k] Steele, 1848 and 1849. [l] R. Paterson, 1848, p. 394. [m] Douglas, 1845, p. 273.

persons who have been starving before the attack, or who have been subjected to lowering treatment. It is usually slight, rarely extends so high as the hips, and seldom lasts longer than two or three weeks.

3. *Gangrene from Pressure* is very rare in relapsing fever, which is not surprising, considering the short duration of the febrile paroxysms.

4. *Gangrene* independent of pressure rarely, if ever, occurs.

5. *Accidental Cutaneous Eruptions.* Perry at Glasgow,[n] and Arrott at Dundee,[o] noted the frequent occurrence of herpetic eruptions around the nose and mouth, especially about the period of relapse.

In a few cases, Cormack observed a pustular eruption around the mouth, immediately after, or simultaneously with, the crisis.[p]

Wardell mentions an instance where several bullæ, containing a sanguineous fluid, appeared over the body. The patient died with uræmic symptoms, and urea was found in the blood.[q]

Another case is mentioned by Douglas, where the fever was followed by an abundant eruption of lichen.[r]

Lastly, boils sometimes break out over the body during convalescence, and may retard recovery (see also page 330).

6. *Inflammatory Swellings or Buboes* are occasionally met with in the relapse, or in convalescence; but, on the whole, they are rare, and do not often give rise to much constitutional disturbance. They may be developed in the parotid, submaxillary, or inguinal region. Wardell records one instance where an inflammatory swelling in the parotid region appeared with the relapse, and was apparently the cause of death.[s]

7. *Effusion into the Joints.* In most cases, the severe articular pains, which occur during convalescence, are unattended by swelling; but there are exceptions. Cormack met with three instances, in which severe pains in the knee-joint were followed by effusion, and with several cases where there was swelling of the ankle-joints.[t] Douglas observed two instances, where the joints of the hand, during convalescence, presented pain, swelling, redness, heat, and stiffness. The attack lasted a few days only. The same writer mentions a third case, where a rigid state of the masseter muscles prevented the movements of the lower jaw; and a fourth, where the same effect was produced by inflammation of the right maxillary articulation, which was tender and presented a circumscribed swelling.[u]

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[n] Perry, 1844, p. 82.
[o] Arrott, 1843, p. 132.
[p] Cormack, 1843, p. 147.
[q] Wardell, 1846, xxxix, 548.
[r] Douglas, 1845, p. 273.
[s] Ibid. xl. 200.
[t] Cormack, 1843, p. 147.
[u] Douglas, 1845, p. 273.

g. Complications referrible to the Uterine System.

1. *Menstruation* may occur at any stage of relapsing fever. At the crisis it is sometimes profuse, and, apparently, critical. Jackson found that severe menstrual discharge took place occasionally, at the invasion of the fever.[x]

2. *Abortions.* A very remarkable feature of relapsing fever is, that pregnant females, no matter at what stage of pregnancy, almost invariably miscarry. This has been a matter of constant observation. For example, of 36 pregnant females who took the fever, under the care of Smith[y] and Jackson,[z] all miscarried but one. The exceptions, indeed, are extremely rare. According to Cormack, abortion occurs most frequently in the relapse; but of 19 cases under Jackson at Leith, 12 aborted during the first paroxysm; 6 during the second; and 1 during the third. Occasionally, it takes place as early as the second day of the fever. Delivery is sometimes followed by copious hæmorrhage, or by rapid sinking and death; but, as a rule, the mother recovers, although, even when pregnancy is advanced, the child is always still-born, or only survives a few hours. This circumstance makes it probable that the abortion is due to the fœtus being poisoned by the maternal blood, aided, perhaps, by the inanition of the mother, before and during the fever.

On the supposition that relapsing fever is but a mild variety of typhus, it would be very remarkable, that, in the former, abortion is almost invariable, and the fœtus dies; whereas, in the latter, abortion is the exception, and when it occurs, the child, if near the full time, usually lives. (See page 197).

Section IX.—Varieties of Relapsing Fever.

Relapsing Fever presents several varieties, according to its degree of severity, or the presence of certain symptoms or complications, such as jaundice, vomiting, cerebral symptoms, hæmorrhages, diarrhœa, or dysentery. The most remarkable and formidable varieties are, on the one hand, that characterized by delirium, stupor, subsultus, coma, or convulsions, or the 'typhoid state'; and, on the other, that which proves fatal by sudden syncope.

Again, there is every possible variety, according to the duration and number of the paroxysms, and the length of the intermissions. As a rule, there are two paroxysms; but, occasionally, there is but one, or there are three; and, in rare cases, there are four or more. In the severer forms, where cerebral symptoms come on at the

[x] Jackson, 1844, p. 423. [y] Smith, 1844, (1), p. 71. [z] *Op. cit.* p. 423.

time of the first crisis, there may be no well-marked intermission, and the paroxysm may appear unusually protracted.

Cormack, in his monograph on the epidemic of 1843, made two varieties of the disease. 1. '*The ordinary, or moderately congestive form,*' characterized by 'bronzing' of the skin, which consisted exclusively of the mild cases, and was never fatal; and 2. '*The highly congestive form,*' the chief characters of which were jaundice, a deep, persistent purple colour of the face, appearing immediately before or after the invasion of the disease, enlarged liver and spleen, and, in some cases, hæmorrhages from the mucous membranes, somnolence, delirium, subsultus, etc.; and, lastly, a remission, rather than an intermission, between the paroxysms. (See page 329).

Section X.—Diagnosis of Relapsing Fever.

The diseases with which relapsing fever is apt to be confounded are:—Typhus, pythogenic or enteric fever, febricula, remittent fever, yellow fever, incipient small-pox, bilious headache, and cerebral diseases.

1. *Typhus.* Prevailing, as they do together, in great epidemics, typhus and relapsing fever have naturally been regarded as varieties of one disease. Yet, in their clinical history, no two diseases can present a greater contrast. The characters, which distinguish relapsing fever from typhus, are mainly the following:

a. The suddenness and severity of the primary rigors (see pages 172 and 349).

b. The absence of that heaviness or stupidity of countenance, so characteristic of typhus (see pages 127 and 329).

c. The much greater frequency of the pulse, as early as the second day of the disease (see pages 135 and 333).

d. The frequent occurrence of an anæmic cardiac murmur, and the absence of the cardiac phenomena, indicative of softening of the left ventricle (see pages 136 and 334).

e. The greater heat of skin, and the absence of the typhus eruption (see pages 127 and 330).

f. The frequency of jaundice, of vomiting, and of tenderness and enlargement of the liver and spleen (see pages 196, 336, and 339).

g. The presence of epistaxis, and other hæmorrhages (see pages 170, and 348).

h. The severe muscular and arthritic pains (see pages 151 and 346).

i. The rarity of delirium, and other cerebral symptoms (see pages 151 and 346).

k. The invariable occurrence of abortion in pregnant females (see pages 197 and 361).

l. The common occurrence of ophthalmia as a sequela (see page 356).

m. The sudden subsidence of the pyrexia, on the fifth or seventh day, accompanied by a copious critical sweat, and followed by apparent convalescence (see pages 177 and 350).

n. After a complete intermission of about a week, the occurrence of a relapse on or about the fourteenth day (see pages 180 and 352).

o. The remarkable difference in the rate of mortality (see pages 218 and 365).

As a rule, the characters of the two diseases are so different, that there can be no difficulty in diagnosis. But, those cases of relapsing fever, in which cerebral symptoms, and especially the 'typhoid state,' are developed, when the patient first comes under observation, may closely resemble typhus, and then, in forming an opinion, we must rely chiefly on the history of the case, the presence or absence of eruption, and the nature of other cases occurring in the same house or family.

2. *Pythogenic* or *Enteric Fever.* (See *Diagnosis of Pythogenic Fever*).

3. *Simple Fever or Febricula.* (See *Diagnosis of Febricula*).

4. *Remittent Fever.* Relapsing Fever, on its appearance in 1843, was regarded by Craigie, Mackenzie, and other observers, as a variety of the remittent fever of tropical countries, and, hence, several of its designations (see page 290). Both diseases commence suddenly, run a short course, have a tendency to relapse, and are often complicated with sickness, jaundice, and hæmorrhages. Tropical remittent fever, however, originates from malaria, affects all classes of the community alike, and is not infectious; whereas relapsing fever occurs in districts free from malaria;[a] and all the circumstances marking its origin and progress oppose the idea of its depending on malaria; it is confined, for the most part, to the poor and destitute, and is infectious. Moreover, there is no resemblance between the intermissions of relapsing fever and the remissions of remittent fever. No form of tropical remittent fever was ever observed, where the febrile paroxysm lasted almost continuously for five or seven days, was then followed by a complete intermission of a week, and afterwards, with tolerable regularity

[a] Only 3 cases of ague were admitted into the Edinburgh Infirmary during the whole epidemic of 1843-4 (*Official Report*, page 2).

on a certain day, by a return of the fever for three or five days. It is true that Craigie, Cormack, and others mention the occurrence of slight irregular remissions in the course of the paroxysms of relapsing fever; but these remissions are far from constant; and from the rigors to the crisis, the paroxysms usually exhibit as continued a course as typhus. Even if they were more common, relapsing fever would not correspond with any form of remittent fever yet described (see also page 302).

5. *Yellow Fever.* The frequency with which relapsing fever is complicated with jaundice, has caused it to be mistaken for true yellow fever. In 1826, Drs. Graves and Stokes[b] published an account of the yellow fever of Dublin, and the 21st chapter of the first volume of Graves's Lectures is entitled 'Yellow Fever of the 'British Islands.' The cases described by these writers appear to have been relapsing fever, complicated with jaundice and cerebral symptoms; and the fact that they differed from true yellow fever was pointed out at the time by O'Brien.[c] The Scotch epidemic of 1843 was likewise regarded as closely allied to, if not identical with, yellow fever, by Cormack, of Edinburgh, Arrott, of Dundee, by several physicians at Glasgow, and by Dr. Graves, of Dublin. In Glasgow, it was even fancied that the disease had been imported by merchant vessels from the West Indies, although, in truth, it had been prevailing on the east coast of Scotland, for some time before it appeared at Glasgow (see page 47).

There is, no doubt, a strong resemblance between the more severe forms of relapsing fever, complicated with jaundice and cerebral symptoms, and true yellow fever. But we have here an illustration of the mistakes which are apt to result from founding analogies or differences between acute specific diseases on symptoms alone, and of neglecting the circumstances under which they appear, or, in other words, their causes. As already remarked, the 'typhoid 'state,' seen in its typical form in true typhus, is not peculiar to that disease, but is liable to be developed in many others. So it is with jaundice, which occasionally appears independently of any mechanical obstruction of the bile-ducts, as a result of other poisons besides that of true yellow fever. Without entering at present into the much-vexed question of the etiology of 'yellow fever,' it may be said to differ from relapsing fever in the following particulars.

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[b] Graves & Stokes, 1826; see also article 'Enteritis,' in *Cyclop. of Pract. Med.* 1833, ii. 59. [c] O'Brien, 1828, p. 532.

a. Yellow fever exhibits no predilection for the poor and destitute, but attacks all classes alike. Indeed, according to some writers, feebleness of constitution prevents rather than favours an attack.[d]

b. Yellow fever attacks the same individual only once; relapsing fever confers no immunity from subsequent attacks.

c. Jaundice is an almost constant symptom in yellow fever, whereas it occurs in less than one-fourth of the cases of relapsing fever.

d. Yellow fever is a most mortal disease; relapsing fever is rarely fatal.

e. Death in yellow fever is usually preceded by 'black vomit,' which, in relapsing fever, even when fatal, is so rare, that some of the most experienced observers have doubted its occurrence.

f. Lastly, the yellow fever of the tropics never follows the peculiar course of relapsing fever—a febrile paroxysm lasting for a week, terminating in a critical sweat, followed by a complete intermission of a week, and then by a second paroxysm. Relapses of any sort are rare in yellow fever.

6. The severe rigors and pain in the back, coupled with headache, vomiting, and quick pulse, may at the onset, lead to the suspicion of *Small Pox*. Although the lumbar pain and vomiting are rarely so severe, as in the early stage of small pox, a diagnosis during the first two days may be difficult, especially if there is any possibility of the patient having been exposed to the poisons of both diseases.

7. The headache is usually less than that of *Dyspeptic or Bilious Headache,* which is also not ushered in by rigors, nor accompanied by the quick pulse and hot skin of relapsing fever.

8. The suddenness of the attack, the rigors, the hot skin, and pains all over the body, as well as in the head, distinguish the onset of relapsing fever from incipient cerebral affections.

Section XI.—Prognosis and Mortality.

As in typhus, the prognosis is based on the rate of mortality, the circumstances influencing that rate, the presence and severity of certain symptoms and complications in individual cases, and the mode of fatal termination.

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[d] Copland's *Med. Dict.* iii. 151.

a. Rate of Mortality.

Relapsing fever is far from being a fatal disease. As compared with typhus or pythogenic fever, its rate of mortality is extremely small. The following table shows the rate of mortality of all the cases admitted into the London Fever Hospital, since 1847.

TABLE XXIV.

Years.	Admissions.	Deaths.	Mortality per cent.
1848	13	1	7·69
1849	29	0	0·
1850	22	2	12·5
1851	256	7	2·73
1852	88	1	1·13
1853	16	0	0·
1854	5	0	0·
1855	1	0	0·
1856	..	..	..
1857	1(?)	0	0·
Total	441	11	2·49
Deducting 2 fatal within two hours after admission	439	9	2·05

Thus, out of the 441 cases only 11 proved fatal, making $2\frac{1}{2}$ per cent., or about 1 in 40, or deducting 2 cases fatal within two hours after admission, the mortality was only 2 per cent. or 1 in 50. This small mortality from relapsing fever has been a matter of general observation. Thus, in the Scotch epidemic of 1843, the mortality according to different observers was as follows:

Locality.	Authority.	Cases.	Death.	Mortality per cent.
Edinburgh . . .	Wardell[e] . .	120	5	4·16
Ditto	Douglas[f] . .	220	19	8·63
Glasgow	McGhie[g] . .	2871	129	4·49
Ditto	Smith[h] . . .	1000	43	4·3
Dundee	Arrott[i] . . .	672	7	1·04
Aberdeen	Kilgour[k] . .	1201	47	3·91
Leith	Jackson[l] . .	216	10	4·63
	Total . . .	6300	260	4·12 or 1 in 24·23

[e] WARDELL, 1846. [f] DOUGLAS, 1845. [g] MCGHIE, 1855. [h] SMITH, 1844.
[i] ARROTT, 1843. [k] KILGOUR, 1844. [l] JACKSON, 1844.

Similar observations have been made since 1843, as shown by the following results:

Locality.	Authority.	Cases.	Deaths.	Mortality per cent.
Edinburgh, 1847-8	Paterson[m] . .	639	20	3·13
Ditto . . do.	Robertson[n] . .	589	23	3·9
Ditto . 1848-9	Official Report .	203	8	3·94
Glasgow since 1843	McGhie[o] . .	4933	276	5 6
Belfast, . 1847-8	Dr. Reid[p] . .	1014	74	7 29
	Total . . .	7378	401	5·43 or 1 in 18·4

Adding all these results to those observed at the London Fever Hospital, we have 14,119 cases, and 672 deaths, or the rate of mortality is 4·75 per cent., or 1 in 21.

b. Circumstances influencing the rate of Mortality.

1. *Times and Seasons.* The mortality, according to season, of the cases admitted into the London Fever Hospital since 1847, is shown in the following table.

TABLE XXV.

Seasons.	Admissions.	Deaths.	Mortality per cent.
Spring . . .	90	3	3·33
Summer . . .	120	1	·83
Autumn . . .	123	5	4·06
Winter . . .	108	2	1·85
Total	441	11	2·49

From this, it would seem, that the mortality is greatest in autumn. The numbers, however, are too small to warrant any general conclusion, and the result was not uniform for each year of the period in question.

As in typhus, the mortality is probably greatest at the commencement of an epidemic. Thus of 22 cases admitted into the London Fever Hospital in 1850, 2, or 12½ per cent. died; of 256 cases in 1851, 7, or 2¾ per cent. died, whereas of 110 cases admitted during the subsequent years, only one died. In the Scotch epidemic of 1843, it was commonly noticed that the cases were most severe and fatal, on the first outbreak of the disease.

[m] R. PATERSON, 1848. [n] ROBERTSON, 1848. [o] MCGHIE, 1855.
[p] *Irish Report*, 1848, viii. 301.

Although in both typhus and relapsing fever, the cases become milder, and the mortality diminishes towards the close of an epidemic; in mixed epidemics of the two fevers, the total rate of mortality has often been noticed to increase progressively as the epidemic advanced. As already stated, this circumstance is due to a gradual increase in the ratio of typhus to relapsing cases.

2. *Sex.* According to the experience of the London Fever Hospital, the mortality among males suffering from relapsing fever is slightly greater than that among females. Of 233 males, 4, or 1·71 per cent., died; and of 208 females, 7 or 3·36 per cent. This result, it may be stated, is opposed to what was found to hold good in typhus, and to what has usually been observed in relapsing fever. Of 215 cases treated by Douglas in 1843, the mortality among the males was $11\frac{1}{2}$ per cent. (14 out of 122), and among the females only $5\frac{1}{3}$ per cent. (5 in 93).[q] Of the cases admitted into the Edinburgh Infirmary, between July 1st, 1843, and October 1st, 1844, when the fever was for the most part relapsing, the mortality among the males was 8·27 per cent. (2,284 cases and 189 deaths) and among the females 7·29 per cent. (2,358 cases, and 172 deaths).[r] In 1847, of 358 males under the care of Robertson, 19, or 5·3 per cent. died, but of 231 females, only 4, or 1·72 per cent.;[s] and of 325 males under Paterson, 15, or 4·61 died, but of 314 females, only 5, or 1·6 per cent.[t] Of the cases admitted into the Edinburgh Infirmary in 1848-9, the mortality was also greater in males (110 cases and 5 deaths) than in females (93 cases and 3 deaths.[u] (See page 220).

3. *Age.* As in typhus, the rate of mortality increases as life advances. Thus, of the admissions into the London Fever Hospital since 1847, the mean age of the fatal cases was much greater than that of those which recovered.

	Number.	Mean Age.
Total cases, in which age known	437	24·41
Cases which recovered	426	24·14
Fatal cases	11	35·09

Table XXVI also shows the rate of mortality at different periods of life.

From this it appears, that, of 304 cases under thirty years of age, only 2 died; whereas, of 133 cases above thirty, 9 died. Similar observations have been made elsewhere. Of 215 cases treated by Douglas in 1843, only 1 out of 135 below thirty died;[x]

[q] Douglas, 1845, p. 273. In 5 of Douglas's cases, which recovered, the sex and age are not stated. [r] *Official Report.* [s] Robertson, 1848.
[t] R. Paterson, 1848, p. 398. [u] *Official Report.* [x] Douglas, 1845, p. 278.

TABLE XXVI.

Age.	No. of Cases.	Deaths.	Mortality per cent.	Age.	No. of Cases.	Deaths.	Mortality per cent.
Under 5 years ...	4	..	..				
From 5 to 10 years	32	..	..	Brought forward..	400	8	..
„ 10 to 15 „	63	1	1·58	From 45 to 50 yrs.	8	2	25·
„ 15 to 20 „	92	1	1·08	„ 50 to 55 „	15	..	..
„ 20 to 25 „	76	..	..	„ 55 to 60 „	7	1	15·28
„ 25 to 30 „	37	..	..	„ 60 to 65 „	5	..	..
„ 30 to 35 „	37	3	8·1	„ 65 to 70 „	1	..	..
„ 35 to 40 „	19	2	10·52	„ 70 to 75 „	1	..	..
„ 40 to 45 „	40	1	2·5	Age not known ..	4	..	..
Carried forward	400	8		Total	441	11	2·49

whereas of 80 above thirty, 18, or 22½ per cent. died; and, of 28 above fifty, 12, or 43 per cent., died.[x] Again, of 153 cases under thirty admitted into the Edinburgh Infirmary in 1848-9, only 3 died, or 2 per cent.; but, of 50 cases above thirty, 5 died, or 10 per cent.; and, of 9 cases above fifty, 3 died, or 33·3 per cent.[y]

4. *Station in Life.* The statistics of the London Fever Hospital furnish little information on this point, as all the cases admitted since 1847, with the exception of 2 (one of which was a doubtful case), were of the poorest class. It has been a common observation in Ireland, that, in epidemics of 'continued fever,' the disease has been more severe and fatal among the rich than among the poor; but, as before stated, the circumstance has been mainly due to the fact, that most of the cases occurring in the upper class have been typhus or pythogenic fever, while a larger proportion of the poor have had relapsing fever.

5. *Recent Residence in a Locality* has little influence on the rate of mortality of relapsing fever. Of 90 patients admitted into the London Fever Hospital, who had resided in London less than six months, 3 died, or 1 in 30; and of 290 patients who had resided more than six months, 8 died, or 1 in 36. This difference was accounted for by the difference in age; the mean age of the fatal cases in the former class being 42·6, and in the latter, 32·25.

6. *Place of Birth and Race.* Of the cases admitted into the London Fever Hospital since 1847, the rate of mortality, according to birth-place, was as follows :—

[x] DOUGLAS, 1845, p. 278. [y] *Official Reports.*

TABLE XXVII.

	No. of Cases.	Deaths.	Mortality per cent.
English - - - - - -	133	6	4·51
Irish - - - - - - -	281	5	1·78
Scotch - - - - - -	2	,,	,,
Foreigners - - - - -	4	,,	,,
Birth-place not noted -	21	,,	,,

From this, it appears that the mortality among the Irish was 1 in 56; among the English, 1 in 22.

7. *The Previous Habits* of the patients influence the progress and mode of termination of the disease. In 6 of Douglas's 19 fatal cases, the health had been greatly impaired by dissipation.

8. There are no data for determining the influence of constitution, previous diseases, mental depression, fatigue and privation, or neglect of treatment on the rate of mortality; but the remarks made on these points, under the head of typhus, are, probably, equally applicable to relapsing fever. Rutty's observation, that 'the poor, abandoned to the use of whey and God's good pro-'vidence, recovered, while those who had generous cordials, and 'great plenty of sack, perished,' has been already accounted for.

c. Prognosis from the Presence of certain Symptoms or Complications.

1. A very rapid pulse, on the first or second day of the disease, is not, as in typhus, a cause of alarm.

2. Profuse perspiration, accompanied by a rapid pulse, is not, as in typhus, a dangerous symptom.

3. Jaundice and minute petechiæ do not, in themselves, indicate danger, unless they be accompanied by cerebral symptoms.

4. Purpura-spots and vibices, however, are only met with in severe cases.

5. Copious hæmorrhages, particularly from the stomach and bowels, are dangerous symptoms.

6. Suppression, or great diminution of the quantity of urine, is usually followed by cerebral symptoms of a dangerous character.

7. Cerebral symptoms, such as stupor, delirium, coma and convulsions, tremors and subsultus, are only observed in the most severe cases, and often terminate in death: even convulsions, however, are not necessarily fatal.

8. It must be borne in mind that fatal syncope, or dangerous cerebral symptoms, occasionally supervene suddenly and unexpectedly.

9. The presence of complications, and especially of peritonitis,

pneumonia, diarrhœa, dysentery, abortion, or erysipelas, always increases the danger.

10. The interval between the paroxysms must not be mistaken for permanent convalescence.

11. After the second crisis, the liability to certain sequelæ, and particularly to severe muscular and arthritic pains, dysentery and ophthalmia must be kept in view. Dysentery supervening during convalescence sometimes terminates fatally.

d. Mode of Fatal Termination.

In fatal cases, death is due to syncope, (page 355), or to uræmic poisoning (page 344), or to some complication, such as dysentery, peritonitis, pneumonia, abortion, hæmorrhages, etc. The fatal event may occur in either paroxysm, in the intermission, or in convalescence. Of 16 fatal cases observed by Douglas, death took place in the primary attack in 4; in the intermission in 1; in the relapse in 5; and during convalescence in 6; in one, death occurred on the 38th day after the accession of the fever from peritonitis, and, in another, as late as the forty-eighth day from dysentery.

Section XII.—Anatomical Lesions.

Relapsing fever being rarely fatal, there are no extensive series of *post-mortem* observations. Still, a sufficient number of examinations have been made, to show that it is characterized by no constant anatomical lesion.

a. Generalities.

Emaciation. The body is usually much emaciated, except when persons in easy circumstances have contracted the disease by direct communication with the sick. The emaciation is due, not so much to the disease, as to previous want.

b. Integuments, Muscles, and Bones.

1. *Discolouration.* Large patches of livid discolouration are often observed on various parts of the body, more particularly on the back, the scrotum and the pinnæ of the ears. In the jaundiced cases, the yellow tint of the skin is often more marked after death than during life.

2. *Spots.* The petechiæ, purpura-spots and vibices, observed during life, persist after death.

3. *The Muscles* do not usually exhibit the dark colour observed in typhus. In one case, dissected by Jenner, they were of a brighter red than natural.

4. *The bones*, and the white tissues of the body generally, are tinged yellow in the jaundiced cases.

c. Organs of Digestion.

1. *The Pharynx and Œsophagus* rarely present any abnormal appearance.

2. *The Stomach.* The mucous membrane is usually perfectly normal, or only slightly injected; but when death has been preceded by urgent vomiting, and more especially, when the rare symptom of 'black vomit' has been present, the lining membrane is much injected, and here and there exhibits patches of ecchymosis and submucous extravasations of blood. Cormack mentions one case where the stomach, over one-third of its surface, was very black, from blood effused on the surface of, and beneath, the mucous membrane. Similar appearances were noticed by Wardell, Douglas, and others, during the Scotch epidemic of 1843. In most cases, the ecchymosed patches do not exceed one or two inches in diameter. The membrane over these patches is softened and lacerable. In rare cases, the stomach contains black blood similar to what has been vomited during life; more commonly it contains only a little yellowish bilious fluid.

3. *The Small Intestines.* In those cases which have been complicated with diarrhœa, the mucous membrane is often more or less injected, particularly towards the lower part of the ileum. And, in rare cases, patches of ecchymosis and of submucous extravasation, similar to those found in the stomach, may be observed. Neither Peyer's patches nor the solitary glands are ever ulcerated, nor do they contain any abnormal deposit; and, indeed, in most cases, the small intestines are in every respect healthy, or only slightly injected.

4. *The Large Intestines* are usually healthy, except in those cases which have been complicated with diarrhœa or dysentery. In the slighter forms of this affection, irregular patches of arborescent and punctiform injection are found scattered irregularly over the surface of the membrane, which, in the vicinity of these patches, is healthy in appearance and consistence. In the more advanced forms, the mucous membrane of the whole of the large intestine, and of the lower two or three feet of the ileum presents the most intense vascular injection, of a deep-red, purple, or dingy-brown colour. The surface also is covered with a pale membranous pellicle, which, here and there, has the appearance of having been separated in patches. Occasionally a few small ulcers, with thickened edges, are found in different parts of the

large intestine.[a] In one case, Cormack found patches of blood extravasated beneath the mucous membrane of the rectum, and altered blood in the fæces.

5. *The Mesenteric Glands* are not enlarged, and present no abnormal appearance.

6. *The Liver*, especially when death occurs during the febrile paroxysms, is usually found enlarged, firm, and loaded with blood; but, even in the jaundiced cases, it exhibits no alteration of structure.

7. *The Gall-Bladder and Bile.* The bile is often dark, thick, and viscid. It has been thought that its inspissated condition might obstruct the ducts and account for the jaundice. But even in the jaundiced cases, the bile-ducts are always perfectly pervious, abundance of bile is found in the duodenum, the fæces are never clay-coloured, and in some cases the bile is even thinner than natural. The lining membrane of the gall-bladder is never ulcerated.

8. *The Pancreas* is normal.

9. *The Spleen* is perhaps of all the internal organs, the one which is most frequently altered. In most cases, it is enlarged and softened, and the enlargement is often greater than that observed in either typhus or pythogenic fever. Jenner, in one instance, found it weigh 38 ounces. It is usually largest when death occurs during the febrile paroxysms; when the fatal event is due to some complication during convalescence, the spleen is often of a normal size. In consistence, the spleen is usually softened, and in some cases diffluent; at other times, it is tolerably firm. Occasionally, pale, red, fibrinous deposits are found in its substance, and near the surface. These deposits are easily broken down, have a finely granular fracture, and are considerably firmer than the surrounding splenic tissue, from which they are separated by a distinct line of demarcation.

10. *The Peritoneum.* Extensive recent peritonitis is occasionally met with (see page 359), usually associated with an inflamed condition of the mucous membrane, but independent of any perforation of the bowel.

d. Organs of Circulation and Blood.

1. *The heart* rarely presents any abnormal appearance. In one case, Cormack observed considerable effusion of blood beneath the endocardium of the left ventricle. The organ has not yet been

[a] Cormack, 1843, p. 49; Douglas, 1845, p. 271.

examined with sufficient care in those cases where death takes place from sudden syncope.

2. *The blood* which is drawn from the body during the febrile paroxysms has frequently been observed to be buffed,[b] although there was no local inflammation. Decolourized fibrinous coagula are found in the heart and large vessels more frequently than in typhus. But, in other cases, and especially in those where hæmorrhages are a prominent symptom, or where cerebral symptoms are present, the blood drawn during life coagulates imperfectly, and after death is found dark and fluid as in typhus.

In several cases, urea has been detected in the blood in considerable quantity.

Microscopic examinations of the blood have shown, that the proportion of white corpuscles is increased, a fact of some interest in connection with the enlargement of the spleen, and the state of anæmia so commonly observed. Dr. Cormack and Professor Allen Thompson in 1843, found the blood in 12 cases to contain an unusually large number of 'colourless globules,' which (as in the first recorded cases of Leukæmia) were mistaken for pus-corpuscles; in some of the cases also, the red corpuscles were very much serrated and notched. Cormack even thought that this condition of the blood could be detected a day or two before the patient was first seized with the fever.[c] Although these observations were called in question by Wardell,[d] all doubt as to their correctness appear to me to have been removed by the details afterwards published.[e] When an opportunity again offers, the blood in relapsing fever ought to be submitted to careful microscopic examination.

e. Organs of Respiration.

1. The *Larynx* and *Trachea* present nothing abnormal.

2. The *Bronchi* are usually healthy, but where catarrhal symptoms have been present during life, they may contain viscid mucus; and their lining membranes may be more or less injected.

3. *The Pleuræ* sometimes exhibit signs of recent inflammation.

4. *The Lungs* not uncommonly present deviations from health; but, on the whole, they are much oftener normal than in typhus. The most common morbid appearances are those of bronchitis.

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[b] Welsh, 1819; Arrott, 1843; Jenner, 1850.
[c] Cormack, 1843, p. 113. [d] Wardell, 1846. [e] Cormack, 1849.

Hypostatic consolidation is occasionally observed; but, in comparison to typhus, is rare. True pneumonia, with a granular appearance on section, is more common than in typhus, and indeed is one of the most common causes of death. Gangrene of the lungs is rare (see page 354).

f. Nervous System.

1. *The Cerebral Membranes* sometimes exhibit increased injection; at other times, they are normal. There is no relation between the amount of vascularity, and the severity of cerebral symptoms during life. In jaundiced cases, the dura mater may be tinged yellow.

2. *The Cerebral Serosity.* An excess of the sub-arachnoid serosity, and of the fluid in the lateral ventricles is occasionally met with. This serosity is colourless or of a pale straw colour; in the jaundiced cases it may be distinctly yellow. In one case, where there had been suppression of urine, followed by cerebral symptoms, during life, Dr. Maclagan found it to contain urea.[f]

3. *The Brain and Cerebellum* exhibit no signs of recent disease. Their substance is of normal consistence, and the number of vascular points may or may not be increased. Occasionally when there is a large quantity of fluid in the ventricles, the surrounding brain-substance is slightly softened. There is no proof that inflammation of the brain, or of its membranes has ever resulted from relapsing fever.

g. Urinary System.

The Kidneys are frequently more or less loaded with blood; but are, in other respects, healthy. No microscopic examination of the renal epithelium has yet been made in those cases where there has been diminution or suppression of urine, with symptoms of uræmic poisoning, but, to the naked eye, the kidneys have appeared normal.

The *post-mortem* appearances of relapsing fever may be summed as follows:

1. There is no specific or constant lesion.

2. The most common lesions are enlargement of the spleen, and probably leukæmia; jaundice, dysentery, enteritis, peritonitis, and pneumonia.

3. Nothing can be discovered in the liver, or in the bile-ducts to account for the jaundice.

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[f] HENDERSON, 1843, p. 223.

4. No lesion can be discovered in the brain or its membranes, even when cerebral symptoms have been most marked.

Section XIII.—Treatment.

The treatment of relapsing fever, like that of typhus, is both prophylactic and remedial.

A. Prophylactic Treatment.

The remarks made on the prophylactic treatment of typhus (page 245) apply also to relapsing fever. Relapsing fever is the appanage of poverty and destitution; and the more completely we succeed in ameliorating the condition of the poor, particularly in times of famine, the more successful shall we be in averting the disease. When an epidemic has broken out, a due supply of nourishment to the poor, attention to ventilation, and the prevention of overcrowding in their dwellings, the providing of baths and public wash-houses, and the timely removal of the sick to hospital are the measures on which we must chiefly rely for arresting its progress. The abolition of the Corn-laws, and the liberal manner in which the English public of the present day respond to appeals in behalf of real distress in any quarter, promise to prevent a recurrence of those frightful epidemics of famine-fever described in former pages.

B. Remedial Treatment.

1.—*Hygienic.*

The rules laid down, when speaking of typhus, as regards the food, drink, and other hygienic measures, ought also to be observed in the case of relapsing fever.

2.—*Therapeutic.*

Before discussing what appears to be the rational treatment, it may be expedient to say a few words respecting certain methods of practice recommended at different times.

1. *Blood-letting.* In the epidemic of relapsing fever in 1817-19, blood-letting was practised largely. The profession was misled by the doctrines of Ploucquet, Clutterbuck, and Beddoes, who taught that there was no such thing as idiopathic fever, but that pyrexia was always dependent on local inflammation; and this error was confirmed by the remarkably small mortality which followed the new method of practice. The fact was lost sight of, that relapsing fever naturally terminates in recovery; and the mortality, after blood-letting in relapsing fever, was compared with the mortality of typhus under the opposite mode of treatment. This is evident,

from the writings of Welsh, the great advocate of blood-letting. Thus, the following extract from his work contains one of the chief arguments in favour of his practice. 'From the registers of 'the Royal Infirmary, it appears, that from January, 1812, to 'January, 1817, 506 fever patients were dismissed cured, or died. 'Of these, 457 were discharged cured, and 49 died, or the propor- 'tion of deaths to recoveries was as 1 in $10\frac{16}{49}$. From the 1st of 'January, 1817, to the 1st of January, 1818, there were 478 fever 'patients dismissed cured, and 33 died; thus the deaths to reco- 'coveries were as 1 in $15\frac{1}{33}$. From the 1st January, 1818, to the '1st of January, 1819, there were 784 patients discharged cured, 'and 41 died, or the deaths were, to the recoveries, as 1 in $20\frac{5}{41}$. '. . . Now, it must be remarked, that it was towards the 'end of the year 1817, that the practice of *free* venesection 'began to be employed in the Royal Infirmary; but it did not 'come into general use till the spring of 1818; and since that 'time the mortality has been steadily diminishing.'[g]

These statements admit of another explanation. Welsh made no distinction between typhus and relapsing fever. The cases during the first of the above periods were mostly typhus; those during the latter were chiefly relapsing fever. The rate of mortality diminished, not from the substitution of venesection for other treatment, but owing to the partial displacement of typhus (a very mortal disease) by relapsing fever, which is rarely fatal. If Welsh, in place of comparing the mortality of relapsing fever, with that of typhus, had compared the mortality among the cases bled under his own care, with that of cases not bled, he might possibly have arrived at a different conclusion. No allusion to a comparison is made in the body of the work; but it appears, from the Tables in the Appendix, that the mortality was much greater in the former class than in the latter. The number of patients under Dr. Welsh, at Queensberry-house, amounted to 743.[h] Of these, during the first paroxysm, 224 were bled from the arm only; 140 were both bled from the arm and leeched; 189 were bled by means of leeches only, and 190 were bled neither generally nor locally. Again, of the 133 patients who suffered a relapse, 42 were bled from the arm during the relapse; 20 were both bled and leeched; 22 were bled by means of leeches only; and 49 were bled neither locally nor generally. 'The total number of ounces of blood drawn during the treatment 'of the cases, both of primary fever and relapse, amounted to '10,166; and the total number of leeches applied amounted to

[g] WELSH, 1819, pp. 169, 170.

[h] Ibid. page 184.

'4,364.'[i] Many of the patients had been also bled before admission into hospital. One patient alone was bled to 100 ounces, and had 26 leeches applied. Now, what was the mortality among the cases that were bled, as compared with that where bleeding was not practised?

Of 364 cases bled from the arm, 20 died, or 1 in $18\frac{1}{5}$.
Of 189 „ leeched, 10 „ „ 1 „ $18\frac{9}{10}$.
Of 190 „ not bled, 4 „ „ 1 „ $47\frac{1}{2}$.[k]

The mortality, therefore, was far more than twice as great among the cases which were bled, as among those which were not bled, although possibly the disease may have been somewhat milder in the latter than in the former.

During the epidemic of 1843, the practice of venesection was tried in several instances; but was almost universally repudiated as worse than useless. Among the benefits ascribed to it in 1818, were, that it frequently cut short the fever: that if it did not at once arrest it, it shortened its duration, by inducing a critical sweat: that it reduced the pulse and the temperature: and that it relieved head-ache and other distressing symptoms. But here again, mistakes arose from confounding relapsing fever with typhus. The short duration, the critical sweat, the sudden fall of the pulse and temperature, with immediate relief to the head-ache, and all the other symptoms, are characteristics of the one disease, although not of the other, but cannot be brought about by blood-letting in either. Speaking of the relief, which in some cases appeared to follow bleeding, Dr. Cormack observes: 'These bene-'ficial changes were often not effects, though sequences of the 'bleeding, as was satisfactorily proved by the very same changes 'frequently occurring as suddenly and unequivocally in patients 'in the same wards, *who were subjected to no treatment whatever.*'[l] 'It is true,' says Wardell, 'that the intense head-ache which there 'was, would be relieved by a full depletion, but this alleviation 'would be only for a brief period, the pulse again rising, and the 'uneasiness and pain complained of becoming as great as ever. 'The copious diaphoresis which invariably determined the critical 'period, lowered the pulse as effectually as blood-letting, and such 'reduction was permanent.'[m] Dr. Jenner, also, after mentioning a case of relapsing fever, which had been bled in the London Fever Hospital, observes: 'Nature, unaided by the loss of blood, in many 'cases effected a much larger improvement in a much shorter

[i] Welsh, page 186.
[k] Ibid. page 184 and table xxii.
[l] Cormack, 1843, p. 151.
[m] Wardell, 1846, xl. 500.

'space of time.'[n] Further evidence tending to the same conclusion, will be found at pages 42 and 256.

It is clear then, that a careful investigation of the question is opposed to the practice of venesection in Relapsing Fever. It is true, that Dr. Christison maintains that the Relapsing Fever of 1843 did not present 'the same strong phlogistic or sthenic cha-'racter' as that of 1817-19.[o] To this it can only be replied, that there is not a single case on record to show that blood-letting cut short the disease, or alleviated the symptoms in 1817-19, in which the improvement could not equally be attributed to the recognized peculiarities of the disease; that the mortality among Welsh's cases was nearly three times as great among the cases bled as among the cases not bled; that other observers of the same epidemic found that the cases did as well, or better, without bleeding (see page 42); and that Dr. Alison stated that the cases which were bled had a slow and unsteady convalescence, in both 1818 and 1843.[p]

2. *Mercury.* In jaundiced cases, several practitioners have recommended mercury in repeated small doses; but there is no proof that it produced any good results. The relief sometimes experienced after its use was probably due to the opium, with which it was always combined.

3. *Remedies for preventing the Relapse.* Various remedies have been tried in the intervals between the paroxysms, with the object of preventing the relapses, but it cannot be said that any of them have proved in the slightest degree effectual. O'Brien, in 1826-7, was inclined to think that quinine might prevent the relapse,[q] but in the Scotch epidemic of 1843, the remedy was tried perseveringly by many practitioners, and found to be perfectly useless.[r] Douglas gave it in 24 cases in doses of from two to four grains three or four times a day. Of the 24 patients, 22 relapsed in hospital, and the remaining 2 were discharged on the fifteenth day, one having all the appearances as if he was about to have a second paroxysm. Moreover, the average date of the relapse was ascertained in 21 cases, and was found to be exactly the same as in the cases treated without quinine.[s] At Edinburgh, in 1847, according to Dr. Paterson,[t] 'much attention was paid, especially towards the beginning 'of the epidemic, to cut short the disease, and to save the patients 'from a relapse. Strict confinement to bed, a strict regulation of 'diet, low diet, common and full diet, quina, bebeerine, arsenic,

[n] JENNER, 1850, xxiii. 31. [o] CHRISTISON, 1858, p. 592. [p] ALISON, 1843, p. 3.
[q] O'BRIEN, 1828, p. 530. [r] CORMACK, 1843, p. 168.
[s] DOUGLAS, 1845, p. 277. [t] PATERSON, 1848, p. 406.

'were all tried in a certain series of cases, but without the least 'effect in warding off the relapse, not even in prolonging its 'recurrence for a single day. It came like a fit of ague, almost 'to an hour.' Dr. Robertson, during the same epidemic at Edinburgh, believed that an emetic given on the fourteenth day, often postponed the relapse for several days, or lessened its violence. He mentions one instance in which it seemed to be deferred by this means for four days. In some cases, however, the relapse does not make its appearance until even a later period than this, when no treatment has been adopted.[u]

Rational Treatment.

It is important to bear in mind, that most cases of relapsing fever recover without treatment of any sort. Rutty, long ago remarked, that 'the poorer sort, abandoned to the use of whey and 'God's good providence, recovered;' and all subsequent experience has confirmed this observation. As in typhus, there is no remedy which can arrest or shorten the disease, and our objects in treatment must be:—1, to promote elimination; 2, to reduce the temperature; 3, to sustain the vital powers; 4, to relieve distressing symptoms; and, 5, to avert and attack local complications.

1. When the patient is seen early in the attack, it may be well to commence with an emetic of ipecacuan and antimony, or of mustard. The act of vomiting unloads the liver, and often affords great relief to the severe pains in the hypochondria. Throughout the febrile paroxysms, constipation is to be counteracted by means of castor-oil, or by five grains of the compound colocynth mass, two grains of blue pill, and three grains of extract of henbane, given at night, and followed, if necessary, in the morning, by two drachms of sulphate of magnesia, in compound infusion of roses. Active purging, however, is to be avoided, and the risk of diarrhœa or dysentery supervening is to kept in view. At the same time, the action of the kidneys is to be kept up by the frequent exhibition of small doses of nitre. Many years ago, Dr. Ross, of Leith, published a paper on the use of nitre, in the relapsing fever of 1818, and spoke of it as 'an invaluable remedy 'for increasing the urine.'[x] In 1843, Dr. Henderson expressed the opinion that head-symptoms might be averted by nitrate of potash, and other saline diuretics.[y] Similar testimony in favour of nitre is borne by Drs. Cormack[z] and Wardell.[a] By keeping up

[u] Robertson, 1848, p. 373. [x] Ross, 1820. [y] Henderson, 1843, p. 222.
[z] Cormack, 1843, p. 161. [a] Wardell, 1846.

the action of the kidneys from the first, we may hope to prevent the occurrence of uræmic intoxication, which is one of the main causes of death in uncomplicated cases. From one to two drachms of nitre are to be dissolved in two pints of barley-water, acidulated with a drachm of dilute nitric acid, and sweetened with a little syrup; this quantity to be taken in twenty-four hours. Acetate of potash and nitric ether may be used for the same purpose; but the nitre has the additional advantage of keeping open the bowels, and so rendering other purgatives unnecessary.

2. We must endeavour to reduce the temperature, by frequent sponging of the surface with cold or tepid water. This practice has the advantages of insuring cleanliness, and of promoting the action of the skin, while it is often very grateful and refreshing to the patient.

3. The vital powers are to be sustained by appropriate food and stimulants, in the administration of which the same rules are applicable as in typhus. In many, perhaps most, cases of relapsing fever, alcoholic stimulants are unnecessary, except during convalescence, and in the stage of languor and exhaustion ensuing on the crisis; but in others, especially where great debility has preceded the attack, they may be required from the first, and even in cases apparently mild, the liability to death from sudden syncope must not be forgotten. The state of the heart ought to be carefully examined, and always, when an anæmic murmur can be discovered, stimulants ought to be given early; it is possible that it is in cases of this nature that sudden syncope is most apt to supervene. When syncope actually threatens, our only hope is in the free use of stimulants. The tendency to death from uræmic intoxication is to be anticipated by the eliminative treatment above recommended, aided by the measures advised for special symptoms.

4. As in typhus, *headache* is one of the first symptoms that the physician is called on to treat. If the administration of an emetic and purgatives, and the local applications to the head, recommended for typhus, afford no relief, and particularly if the pain prevents sleep, recourse should be had to opium, which not only procures sleep, but usually causes great abatement of the pain. Opium is also the best and surest remedy for the *muscular and arthritic pains*, which are often the source of intense distress. For these purposes, it may be advantageously combined with antimony, unless the patient suffers at the same time from sickness (see p. 275).

Vomiting and pain and tenderness in the hepatic and splenic regions are often greatly relieved, by the exhibition of an emetic, by

clearing out the bowels, and by dry cupping, or the application of warm fomentations, sinapisms, turpentine-stupes, or blisters over the seat of pain. If, notwithstanding these measures, the sickness or pain continues, opium is again the remedy. It may be given by the mouth in the solid form, or five minims of Battley's solution, in an effervescing mixture, may be repeated every three or four hours; but the best mode of administration is by the rectum. At the same time, great relief is sometimes derived from repeated doses of lime water, and from sucking small pieces of ice. Hydrocyanic acid, creasote, and other remedies, have been tried for the sickness, and have been sometimes found useful; but all are inferior in efficacy to opium, which is, in fact, a most invaluable remedy in the treatment of relapsing fever.

The mineral acids are now generally considered the best remedies in cases of jaundice depending on blood-poisoning;[b] and, accordingly, when *jaundice* appears in relapsing fever, it may be well to give nitro-hydrochloric acid in combination with nitre, as recommended at page 266. It must be remembered that the danger lies in the contamination of the blood with urinary products, and not with bile. Mercury is of no use in such cases. Dr. Corrigan states, that turpentine, in does of from one to two drachms three times a day for a few days is usually efficient in removing the jaundice.[c]

In all cases of relapsing fever, particular attention must be paid to the state of the *urine*, and especially about the time of the first crisis. When it is much reduced, or entirely suppressed, and particularly when the patient is at the same time stupid, confused, or drowsy, the bowels are to be freely moved by compound jalap powder, or by a turpentine enema. Determination to the skin is to be promoted by the hot-air bath, or by the hot wet blanket, already recommended when speaking of typhus. Dry cupping, sinapisms, and the wet compress (see page 279) may be applied over the loins, while, at the same time, ten grains of nitre, or some other saline diuretic, are to be given every two or three hours.

Delirium and other *cerebral symptoms*, in relapsing fever, are to be treated on the same general principles as recommended for typhus.

5. The principal *complications* and *sequelæ*, which call for treatment in relapsing fever, are pneumonia and bronchitis, muscular and arthritic pains, œdema, diarrhœa and dysentery, peritonitis, enlargement of the spleen and ophthalmia.

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[b] Frerichs, *Klinik der Leberkrankheiten*, Syd. Soc. Transl. i. 236.

[c] Corrigan, 1853, p. 90.

Pneumonia and *bronchitis* require the same treatment as in typhus (see page 281).

Various remedies have been given for the severe *muscular and arthritic pains* occurring during convalescence. Tweedie strongly recommends small doses of extract of colchicum with calomel and Dover's Powder.[d] Cormack, however, gave colchicum, both in large and small doses, an extensive trial, and came to the conclusion that it was of little or no use. He also tried the iodide of potassium, which he fancied sometimes to afford a little ease.[e] But the remedies, on which most reliance is to be placed, are tonics, such as quinine and iron, and opium. The opium is to be given internally, and may also be applied externally in the form of liniment or fomentation.

Œdema of the lower extremities is best treated with steel and mineral acids, a nutritious diet, and bandaging the legs.

Small doses of laudanum in decoction of logwood, or an astringent mixture containing kino or catechu, with opium, will, in most cases, check *diarrhœa*. But when there is tenesmus, with bloody and slimy motions, indicative of *dysentery*, the best remedies are ipecacuan and opium. The ipecacuan may be given in the form of 'Twining's pill,' which has long enjoyed great repute for the treatment of dysentery in India, and in which it is combined with blue-pill and extract of gentian, or it may be prescribed as follows:—

℞—Pulv. Ipecac. . . . . gr. ij.
Pulv. Ipecac. Co. . . . gr. v.
Hydrarg. c. Creta . . . gr. iij Misce.
Fiat. pulv. quater in die sumend.

Or,

℞—Pulv. Ipecac. . . . . gr. iij.
Pulv. Acaciæ . . . . gr. v. Misce.
Fiat. pulv. 4tâ q.q. horâ sumend.

An enema of starch and opium ought also to be administered from time to time, especially when there is much tenesmus, and occasional doses of castor oil are useful, if the stools are scanty, and the abdomen distended. Warm fomentations are to be applied over the abdomen, and the diet is to be restricted to articles which are nutritious, but non-irritating, such as milk, farinaceous food, eggs, etc. If the dysentery assume a chronic form, the mineral astringents, such as the sulphate of copper, the acetate of lead, and the nitrate of silver, in combination with small doses of opium, ought to be substituted for the ipecacuan.

In cases of *peritonitis*, large and repeated doses of opium (gr. j.

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[d] TWEEDIE, 1860, p. 592. [e] CORMACK, 1843, p. 164.

every hour), fomentation of the abdomen and absolute rest are the only remedies likely to be of any benefit.

When *enlargement of the spleen* persists during convalescence, a combination of sulphate of iron and quinine internally, and the external application of iodine, are the measures most likely to be of service.

From the observations made on the *post-febrile ophthalmia,*[f] different plans of treatment appear indicated according to the stage of the malady. In the simply amaurotic condition, tonics, such as quinine and iron, are evidently called for. By such remedies, with a liberal diet, and blisters behind the ears, we may hope to avert the inflammatory stage. As soon as this stage shows itself, a few leeches are to be applied to the temples, and a powder containing one grain of calomel, one or two grains of quinine, and a quarter of a grain of opium, with a little sugar, may be given every four or six hours. When the gums become affected, the quinine is to be continued without the calomel. At the same time, the pupils are to be kept dilated by dropping occasionally within the eyelids a solution of belladonna or atropine, and the leeches are to be followed by blisters behind the ears, which should be kept open for some time. These remedies should be combined with a nutritious diet.

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[f] See references at page 356.

CHAPTER IV.

Pythogenic or Enteric Fever.

Sect. I.—Definition of the Disease.

An endemic, communicable disease, generated and probably propagated by certain forms of decomposing organic matter. Its symptoms are: a commencement, often insidious, or marked by slight rigors, a sensation of chilliness, or profuse diarrhœa; pulse usually frequent and soft, but variable in the same patient; febrile symptoms in mild cases often presenting a remittent character; tongue often red and fissured, occasionally becoming dry and brownish; in most cases, but not invariably, increased splenic dulness, tympanitis, abdominal tenderness, gurgling in the iliac fossæ, and diarrhœa, with or without melœna; skin warm, with occasional sweats; an eruption of isolated, elevated, rose-coloured spots, vanishing on pressure, first appearing between the seventh and fourteenth days, and coming out in successive crops, each of which lasts two or more days; frequently epistaxis; prostration coming on late, and often slight; head-ache, sometimes followed by stupor and active delirium; but mind often clear throughout the attack, even in fatal cases; dilated pupils; the disease often protracted to the thirtieth day, and occasionally, though rarely, followed by a relapse of all the symptoms, including the eruption; after death, disease of the solitary and aggregated glands of the ileum, and enlargement of mesenteric glands.

Sect. II.—Nomenclature.

1.—*Synonyms derived from its supposed resemblance to Typhus.*

Typhus Nervosus (*Sauvages*, 1759); Typhus mitior and Synochus *pro parte* (*Cullen*, 1769); Abdominal Typhus and Darm-typhus (*Autenrieth*, 1822, and *German Writers generally*); Synochus and Typhus with Abdominal Affection (*Southwood Smith*, 1830); Fièvre Typhoïde (*Louis*, 1829; *Chomel*, 1834); Typhus Gangliaris vel Entericus (*Ebel*, 1836; *Schönlein*, 1839); Typhoid Fever (*Stewart*, 1840; *Bartlett*, 1842; *Jenner*, 1849); Mild Typhoid Fever (*Copland*, 1844); Ileo-typhus (*Griesinger*, 1857); Typhia (*Farr*, 1859); Typhus (*many writers*).

2.—*From its Mode of Prevalence.*

Febris non-pestilens (*Forestus*, 1591); Endemic Fever (*many writers*); Autumnal or Fall Fever (*Flint*, 1852; and *American Writers generally*).

3.—*From its Remittent Character.*

Πυρέτος ἡμιτριταῖος? (*Hippoc.*); Hemitritæus? Tritœophyas? and Triphodes? (*auctor. antiq. var.*); Febris semitertiana seu composita (*Galen? Forestus*, 1591; *Spigelius*, 1624); Tritœophya typhodes (*Mangetus*, 1695); Remittent Fever (*T. Sutton*, 1806); Infantile Remittent Fever (*Evanson* and *Maunsell*, 1836; *and many writers*).

4.—*From its Lengthened Duration.*

Febris lenta (*Forestus*, 1591; *Willis*, 1659; *Linnæus*, 1763; *Vogel*, 1764); Slow Fever (*Strother*, 1729; *Langrish*, 1735); Febris chronica? (*Juncker*, 1736); Common Continued Fever (*Armstrong*, 1816); Fièvre Continue (*Lerminier and Andral*, 1823).

5.—*From its supposed Nervous or Hysteric Character.*

Nervous Fever (*Gilchrist*, 1734); Slow Nervous Fever (*Huxham*, 1739); Febricula, or Little Fever, commonly called the Nervous or Hysteric Fever, Fever on the Spirits, Vapours, etc. (*Manningham*, 1746); Irregular Low Nervous Fever (*Fordyce*, 1791); Nervenfieber (*German Writers*); Fièvre Nerveuse (*French Writers*); Low Fever (*many writers*).

6.—*From the occurrence of Putrid or Septic Symptoms.*

Febris putrida (*Riverius*, 1623); Febris putrida quæ vulgo *lenta* appellatur (*Willis*, 1659); Febris putrida nervosa? (*Wintringham*, 1752); Febris putrida aut biliosa (*Tissot*, 1758); Febris a putredine orta (*A. Tralliani, quoted by Burserius as Syn. for his Feb. gastric. ac.*, 1785); Febris atacta, *pro parte* (*Selle*, 1770); Fièvre ataxique, *pro parte*, and F. adéno-meningée (*Pinel*, 1798); Entérite Septicémque (*Piorry*, 1841); Sepimia (*Hare*, 1853).

7.—*From its resemblance to Hectic Fever.*

Febris hectica (*Willis*, 1667); Infantile hectic fever (*various writers*).

8.—*From the absence of the true Typhus Eruption.*

Febris petechizans vel spuria (*Hoffmann*, 1699).

9.—*From the common occurrence of Gastric Derangement, Bilious Vomiting, etc.*

Febris gastrica (*Ballonius*, 1640); Febris acuta stomachica aut intestinalis (*Heister*, 1736); Febris glutinosa gastrica (*Sarcone*, 1765); Febris gastrica acuta (*Burser.*, 1785); Fièvre méningo-gastrique (*Pinel*, 1798); Gastrisches Fieber (*Richter*, 1813); Fièvre gastrique (*Dict. des Sc. Méd.*, 1816); Epidemic Gastric Fever (*Cheyne*, 1833); Gastric Fever (*Craigie*, 1837); Febris biliosa (*Galen? River.* 1623; *Stahl*, 1700; *Juncker*, 1736); Bilious Fever (*Pringle*, 1750; *Rutty*, 1770); Febris biliosa putrida (*Selle*, 1770); Febbre biliosa (*Benelli*, 1775); Synochus biliosus (*Sauvages*, 1759); Bilio-gastric Fever (*Copland*, 1844); Gastro-bilious, and Bilious Continued Fever (*Modern Writers*).

10.—*From the Intestinal Symptoms and Lesions.*[g]

Febris Colliquativa? (*J. R. Fortis*, 1668); Febris Stercoralis? (*Quesnay*, 1753); Febris Mucosa (*Selle*, 1770); Febris Pituitosa (*Stoll*, 1785; *Strack*, 1789); Febris Colliquativa primaria seu essentialis (*Burserius*, 1785); Morbus Bilioso-Mucosus (*Knaus*, 1786); Febris Pituitosa Nervosa (*Jacobi*, 1793); Schleimfieber (*Kanz*, 1795); Fièvre Muqueuse (*French Writers*); Mucous or Pituitous Fever (*Copland*, 1844).

Febris Mesenterica Maligna (*Baglivi*, 1696; *Hoffmann*, 1699); Febris Intestinalis vel Mesenterica (*Riedel*, 1748); Febris Mesenterica acuta (*Burchard, quoted by Burserius*, 1785); Fièvre entéro-mesentérique (*Petit and Serres*, 1813); Enteritic Fever (*Mills*, 1813); Gastro-entérite (*Broussais*, 1816); Entero-mesenteric Fever (*Abercrombie*, 1820); Febris Mesaraica (*Wendt*, 1822); Dothiénentérite (*Bretonneau*, 1826; *Leuret*, 1828; *Christison*, 1840); Muco-enteritis (*various writers*); Fever, with Affection of the Abdomen (*Alison*, 1827); Fever, with Ulceration of the Intestines (*Bright*, 1829); Gastro-enteric and Gastro-splenic Fever (*Craigie*, 1837); Entérite-folliculeuse (*Cruveilhier*, 1835; *Forget*, 1841); Enteric Fever (*Ritchie*, 1846; *Wood*, 1848; *W. T. Gairdner*, 1859; *Tweedie*, 1860); Febris Tympanica (*Babington*, 1853); Intestinal Fever (*W. Budd*, 1856).

11.—*From its supposed dependence on Worms.*

Typhus Hysterico-verminosus (*Sauvages*, 1759); Febris Verminosa (*Selle*, 1770); Worm Fever (*various writers*).

12.—*From its Mode of Origin.*

Night-soil Fever (*Brown*, 1855); Pythogenic Fever (*Murchison*, 1858); Cess-pool Fever (*var.*).

13.—*Other Designation.*

Miliary Fever (*Pringle and De Haen*, 1760).

The term *typhoid*, commonly applied to this fever, is, in many respects, inappropriate. In the first place, it literally means *like typhus*, and consequently, it is at variance with all precedent in the accepted nomenclature of species in science. Secondly, it is constantly employed in an adjective sense, to designate a group of symptoms, which may appear in the course of any disease; and thirdly, a large proportion of the cases of so called 'typhoid fever' exhibit no symptoms of a typhoid or typhus-like character. It follows, that the use of the term typhoid, to designate a specific fever, tends to create confusion; and, indeed, it is probable, that this very name has contributed to make many regard the affection in question, as merely a variety of typhus. At the same time, it

[g] Many of the cases described by Cullen and his successors, as 'Enteritis Erysipelatosa,' were probably examples of this fever. (See description of it by Alison, 1844 (No. 2), p. 323.)

may be doubted, if any of the numerous synonyms by which the disease has been known, be more appropriate. For example, I am inclined to question the propriety of employing a name derived from the abdominal lesion, as most such designations are calculated to revive the exploded doctrines of Broussais. Even the term *Enteric Fever*, adopted by several recent writers, and perhaps the least objectionable, is apt to convey the erroneous impression, that the fever is the result of the intestinal lesion. These considerations induced me to suggest a few years ago, the name *Pythogenic Fever* derived from what I endeavoured to show was the cause of the fever (πύθογενης from πύθων (πύθομαι, putresco) and γεννάω). The reception, which this name has met with by the profession, has encouraged me to adopt it in this work.

Section III.—Historical Account.

Some of the descriptions of the Greek writers probably referred to pythogenic fever. Hippocrates states, that in the course of two successive autumns, he met with many cases of fever of the continual type, characterized by diarrhœa, offensive watery stools, bilious vomiting, tympanitis, abdominal pain, 'red rashes,' epistaxis, sleeplessness, or a tendency to coma, delirium, and subsultus, irregular remissions, a lengthened duration, and great emaciation.[h]

Galen's *Hemitritæus*, which was thought to be produced by the grafting of a tertian on a quotidian intermittent, and particularly that variety designated bilious fever, (χολοδὸς πυρετὸς), was probably the same disease.[i] But whatever was the nature of the cases referred to by Galen, there can be little doubt that the *Hemitritæus* or *Febris Semitertiana* of later writers was true pythogenic fever. Spigelius speaks of this fever as common in various parts of Italy in the early part of the seventeenth century. Among the symptoms, he mentions abdominal pain and tenderness, bilious vomiting, urgent diarrhœa, and sometimes melœna, sleeplessness or lethargy, delirium, irregular remissions, no marked crisis, and occasional relapses. The *post-mortem* appearances, moreover, are said to have consisted in inflammation, and sometimes gangrene and sphacelus of the small and large intestines. The accounts of several autopsies are given in his work. Of one, he says, 'In dissecto cadavere reperta sunt intestina tenuia in-

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[h] *De Epid.* lib. i. Syd. Soc. Transl. i. 354-9 and 420.

[i] Galen, *Op. Om.* ed. Basil, v. 362; ed. C. G. Kühn, Lipsiæ, 1824, vii. 350; Paulus Ægineta, Syd. Soc. Transl. vol. i.; Celsus, lib. iii. 8.

'flammata; ileum qua colo et cœco adhœrebat, sphacelatum:' in another, 'In eo tenuia intestina inflammata vidimus: et ilei portio-'nem magnam versus colon sphacelatam.' He maintained that the fever was not symptomatic of the local inflammation, but depended on a putrid substance in the veins. As to treatment, he recommended copious venesection, antimony, warm fomentations and clysters.[k]

Not long after, similar observations were made by Panarolus and Thomas Bartholin. According to Panarolus, many cases of fever proved fatal at Rome in 1694, and on dissection, the intestines 'apparebant tanquam exusta.'[l] Now various writers have compared the yellow sloughs often found adherent to Peyer's patches in pythogenic fever, to the superficial eschars resulting from the application of the actual cautery to a mucous surface.[m]

About the same time, Willis, in England, described a fever which differed from the *febris pestilens* (typhus), in being less contagious, in the absence of eruption, in its longer duration, in the imperfect crisis, and in its tendency to produce local complications. He alluded particularly to a dysenteric form of fever accompanied by pustules and ulcers in the small intestines, which he compared to the external pustules of variola, an idea which French writers long afterwards claimed the credit of originating.[n]

Sydenham also described a fever as distinct from the *febris pestilens*. It varied greatly in its severity, lasted from fourteen to thirty days, and was characterized by great tendency to diarrhœa and vomiting, by delirium, epistaxis, etc. Purgatives were always injurious. He recommended bleeding and emetics at the commencement; enemata of milk and sugar, opium to check the diarrhœa, and wine, when the fever assumed a hectic character.[o]

Baglivi, of Rome, in the latter part of the seventeenth century, described the *hemitritæus* of previous writers under the title of *Febris Mesenterica*, and maintained that it was always accompanied by, and depended on, inflammation of the intestines and enlargement of the mesenteric glands. It was of an irregularly remittent character, but was influenced by critical days, although, in most cases, it lasted from fourteen to twenty-one days. In some cases, he observed, there was scarcely any fever, when, all at once, the patient died from inflammation of the viscera. He recommended moderate venesection, baths, warm fomentations to the abdomen, and, above all, patience. Wine and bark, which he thought of

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[k] SPIGELIUS, 1624. [l] PANAROLUS, 1654, pent. iv. obs. 8. [m] RITCHIE, 1855, p. 262.
[n] WILLIS, 1659, ed. 1682, p. 86. [o] SYDENHAM, 1685, ed. 1844, lib. i. 4, p. 39.

great service in *febris pestilens* (typhus), he believed to be highly injurious in mesenteric fever, and he added:—'Fuge purgantia tanquam pestem.'[p]

Lancisi, who wrote soon afterwards on the fevers of Rome, observed, that, on opening the bodies of certain patients who had died of semitertian fever, wounds were found in the intestines, which, in some instances, had perforated all the coats. The ulcers were spoken of as wounds, because they were thought to be produced by intestinal worms. According to Lancisi, semitertian fever differed from the *febris castrensis* (or typhus) in the presence of lumbrici, which irritated and wounded the intestines.[q]

In 1699, F. Hoffmann, of Halle, described the semitertian fever as accompanied by abdominal pain, vomiting and purging, and sometimes by delirium. After death, he says, gangrene and sloughing of the small intestines were found. The same writer mentions another fever under the title of *Febris petechizans vel spuria*, which he distinguished from the *Febris petechialis vera* (or true typhus). This was also probably pythogenic fever. It was characterized by an insidious commencement, vomiting and purging, and by the appearance, about the seventh day, of an eruption on the trunk, consisting of elevated papules, which disappeared completely upon pressure. In both the semitertian fever and the febris petechizans, depletion was considered beneficial, and stimulants injurious.[r]

Strother, in his account of the epidemic of typhus at London in 1727-29, distinguished it from the *slow fevers*, which were of a somewhat remittent character, and which were dangerous on account of their insidious commencement, and of their proving suddenly fatal. One form of this fever, viz., 'the Lent Fever, is 'a symptomatical Fever, arising from an inflammation, or an 'ulcer, fixed on some of the bowels.' The spleen and liver were usually enlarged. Bleeding he regarded as 'the adequate cure.'[s]

In 1734, Dr. Ebenezer Gilchrist, of Dumfries, N.B., published an '*Essay on Nervous Fever*.' This description evidently refers to pythogenic fever. Thus he speaks of its long duration, and of its frequent occurrence in children. The symptoms varied greatly in different cases, but among the most common were diarrhœa, abdominal pain, melœna, epistaxis, partial sweats, which gave no relief; and, in the advanced stages, delirium and other cerebral

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[p] Baglivi, 1696, ed. 1704, p. 51.
[q] Lancisi, 1718, pp. 55, 57.
[r] Hoffmann, 1699, ed. 1740, vol. ii. cap. 5, p. 40, and cap. 10, p. 75.
[s] Strother, 1729, pp. 15, 164.

symptoms. He observes:—'I take this fever to be very different 'in its nature and changes from other fevers' prevalent in Scotland.[t]

In the following year, Dr. Browne Langrish, of London, drew a similar distinction between the '*Slow Nervous Fevers*' and the '*Malignant Continued Fever*,' the former being characterized by a quick but variable pulse, vomiting, purging, and a duration of from twenty to thirty days. In the treatment of nervous fever, Langrish condemned both bleeding and purging.[u]

Four years later, Huxham published the first edition of his 'Essay on Fevers,' in which he devoted a chapter to the differences between the '*Slow, Nervous Fever*,' and the '*Putrid, Malignant, 'Petechial Fever*.' His descriptions leave little doubt, that by the former title he referred to pythogenic fever, and by the latter, to typhus. He observes, 'I cannot conclude this Essay on Fevers, 'without taking notice of the very great difference there is between 'the *putrid malignant* and the *slow, nervous fever;* the want of 'which distinction, I am fully persuaded, hath often been pro-'ductive of no small errors in practice, as they resemble one 'another in some respects, though very essentially different in 'others.'[x]

In 1746, Sir Richard Manningham, F.R.S., gave an excellent description of Pythogenic Fever, under the title of '*Febricula, or 'Little Fever*.' This fever, he said, was popularly designated the '*Nervous or Hysteric Fever, Low Continued Fever, Fever on the 'Spirits, Vapours, Hypo, or Spleen*.' Among the symptoms were, a red, often dry tongue, abdominal pains, diarrhœa, hæmorrhages, quick but variable pulse, loss of memory; and, in a few cases, slight delirium. He dwelt particularly on its insidious origin, and said, that at the beginning it was apt to be disregarded, 'till, 'at length, more conspicuous and very terrible symptoms arise, 'upon which the physician is sent for in the greatest hurry, and 'the little, neglected fever proves of very difficult and uncertain 'cure, and too often becomes fatal in the end.' He condemned the practice of bleeding, and recommended cordials and diaphoretics.[y]

Not long after, a discussion arose between Sir John Pringle and Professor De Haen, of Vienna, as regards the treatment of fever. De Haen advocated the necessity of blood-letting, whereas Pringle observed that 'many recovered without bleeding, but few who 'had lost much blood,'[s] and recommended stimulants. It turned

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[t] GILCHRIST, 1734, p. 347. [u] LANGRISH, 1735, p. 343. [x] HUXHAM, 1749. [y] MANNINGHAM, 1746.

out, however, that these two observers were dealing with different diseases. Pringle's malignant fever of the hospital and jail, with petechiæ, was typhus; whereas De Haen's petechial and miliary fevers were, for the most part, pythogenic. Pringle gives the most unmistakeable description of the eruption of typhus; but the eruption in De Haen's fevers is described as consisting of isolated round elevated spots, which came out in successive crops, and which were occasionally interspersed with true petechiæ and vibices. This difference was pointed out by Pringle, who, in his reply to De Haen's attack, observed that one great cause of confusion was the undefined meaning of the term *petechiæ*, and added: 'I have 'never considered the gaol or hospital fever and the miliary fever 'as similar, and indeed, I may venture to say, that, as the symp- 'toms of the two are so much unlike, they ought to be treated as 'different in species,' and again: 'The *miliary fever* is incident to 'all ranks of people, living in the best air and in the most cleanly 'manner, whereas the *malignant fever*, which I treat of, is scarce 'to be seen but among the lowest people crowded together in close 'and foul places, such as in military hospitals, jails, and transport- 'ships.'[z]

De Haen's miliary fever was described by his successor Stoll, under the designations of *pituitous* and *slow nervous fever*. Stoll relates the case of a boy who died on the fourteenth day of this fever. His symptoms, during life, had been vomiting, diarrhœa, and colicky pains, associated with general fever; but this was so slight, that until the twelfth day, he was able to walk to the hospital for medicine. After death, the small intestines were found inflamed and gangrenous, and the mesenteric glands enlarged, and near the lower end of the ileum there was a perforation.[a]

Many other accounts of pythogenic fever on the Continent were published during the eighteenth century. Riedel described a *febris intestinalis*, in which the lower portion of the ileum was found gangrenous after death.[b] The same fever is also reported as very prevalent at Stuttgart, in 1783;[c] at Göttingen, in 1785; and at Hildesheim, in 1789.[d] Not a few writers pointed out the difference between it and typhus. Burserius, for example, after describing with tolerable accuracy, the symptoms and *post-mortem* appearances of pythogenic fever, added, that although it sometimes

[z] Pringle, 1750; 4th ed. 1764, app. pp. 99, 101; De Haen, 1760; Ritchie, 1855, p. 264; Jenner, 1853, p. 416.
[a] Stoll, *Rat. Med.* ii. 407; Ritchie, 1855, p. 265.
[b] Riedel, 1748, p. 45. [c] Cless, 1837. [d] Duncan's *Annals of Med.* 1796, i. 73.

simulated petechial fever, 'multum discrepare videtur.'[e] In 1760-1, an epidemic of fever occurred at Göttingen, which has acquired some notoriety, and requires particular notice. It was described by Roederer and Wagler, under the name of *morbus mucosus*, and has been regarded by most succeeding writers, as identical with the pituitous fever of Stoll, and the typhoid fever of modern times. But, after carefully reading the original monograph, I am inclined to doubt the correctness of such a view, and to believe, that the fever referred to, was probably for the most part typhus complicated with dysentery. The disease broke out in a crowded, famished garrison, during a siege, in November, 1760. Roederer and Wagler regarded it as a degenerated form of dysentery, with which the garrison had been afflicted for three months before. Although the intestines were found after death ulcerated and gangrenous, these lesions were always in the large intestines. In thirteen cases, the *post mortem* appearances are described with great minuteness, but in none was the ileum ulcerated; while, in the general observations on the anatomical lesions, it is remarked concerning the small intestines: 'Tunica interna, 'licet inflammata, tamen continua est.' The enlargement of the mucous follicles figured and described, were observed in the stomach, duodenum and colon, and in only one case is any mention made of enlargement of the agminated and solitary glands of the ileum.[f]

Meanwhile in England, the distinctions drawn by Gilchrist and Huxham, between the slow nervous fever, and the malignant fever of the hospital, were not lost sight of, and were exciting some discussion. Dr. Vaughan, of Leicester, in a letter addressed to Dr. Lettsom, speaks of the *febris nervosa* as 'a very different 'disease to the *febris carcerum*, in its attack, progress, termination, 'and cure,' and blames Cullen for not distinguishing them;'[g] and Dr. Erasmus Darwin, of Derby, in another letter addressed to Dr. Lettsom, in 1787, proposes as a question for discussion at the Medical Society: 'Whether the nervous fever of Huxham be 'the same as the petechial or jail fever;'[h] while Dr. Willan, in 1799, observed that Cullen had 'improperly comprised under the 'term typhus, the slow or nervous fever described by Gilchrist and 'Huxham, which may rather be considered as a species of hectic, 'and is not received by infection.'[i] The intestinal lesions of the slow nervous fever also began to be noted. They did not escape

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[e] BURSERIUS, 1785, p. 449. [f] ROEDERER and WAGLER, 1762, pp. 4, 8, 19, 179.
[g] *Life of Dr. Lettsom*, by Pettigrew, iii. 161-2. [h] Ibid. iii. 118.
[i] WILLAN, 1801, p. 231.

the notice of John Hunter, as is shown by two preparations in his museum at the Royal College of Surgeons;[k] and in 1799, one of Hunter's preparations was figured by Matthew Baillie.[l]

There is also evidence to show, that during this century, pythogenic fever was not unknown in Ireland. Rutty makes frequent mention of a continued fever at Dublin, which prevailed, for the most part, in autumn, was protracted to three or four weeks or upwards, and was accompanied by diarrhœa and hæmorrhages.[m] Dr. Macbride, of Dublin, in 1772, spoke of the *febris nervosa* (a protracted fever, attended by diarrhœa), as a different species from the *putrid continual fever*, which was contagious, and accompanied by a florid eruption, gradually passing into petechiæ.[n] Lastly, Dr. Sims, in his account of the epidemic of typhus in Tyrone, in 1771, remarked, that it was different from the nervous fever of Gilchrist and Huxham, although he believed that nervous fever, in its advanced stage, might degenerate into malignant hospital fever, so that it was impossible to distinguish them.[o]

With the commencement of the present century, the pathological anatomy of fever began to be carefully investigated in France. M. Prost, of Paris, in 1804, announced that 'les fièvres muqueuses, 'gastriques, ataxiques, adynamiques, ont leur siège dans la mem-'brane muqueuse des intestins' (vol. i., p. 23). He stated that he had dissected the bodies of upwards of 200 patients, who had died of fever in Paris, and that he had invariably found the intestines inflamed (p. 56); and, he added, what is now known to be erroneous, that this inflammation was always in proportion to the severity of the delirium and other febrile symptoms (p. 57). Prost erred in mistaking every *post-mortem* redness of the intestinal canal for inflammation; and although he described correctly the ulcerations peculiar to pythogenic fever, he regarded them as merely the ultimate stage of ordinary inflammation, and was unacquainted with the peculiar seat and nature of the disease. Of the 113 *post-mortem* examinations of different diseases recorded in his work, 16 only appear to have been well-marked examples of pythogenic fever.[p]

Broussais did little more than extend the views advocated by

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[k] *Pathol. Catal.* Nos. 1214 and 1219.

[l] *Plates of Morbid Anatomy*, fasc. 4, pl. ii. fig. 3. Dr. William Stark of London, has often been referred to, as the first to localize the lesions of enteric fever in the intestinal glands; but his drawings, and still more the clinical account of the cases from which they were taken, leave no doubt in my mind that the disease which he described, was not enteric fever. STARK'S *Works*, 4to. Lond. 1788, pp. 5, 7. [m] RUTTY, 1770, pp. 51, 181, 187, 202, 250, &c. [n] P. 336.

[o] SIMS, 1773. [p] PROST, 1804.

Prost. He was aware, that the ulcerations found in fever frequently had their seat in the intestinal glands, but he thought it useless to distinguish between this form and inflammations of other portions of the intestine. So little did Broussais appreciate the nature of the lesions of pythogenic fever, that, in describing this disease as the type of his 'gastro-enterite,' he maintained, that in variola, measles, and scarlet fever, death was due to the same 'gastro-enterite.' Believing that its symptoms were the result of inflammation, Broussais was the advocate of copious depletion, and his writings have more or less influenced the practice of many continental physicians to the present day.[q] In 1813, pythogenic fever was described with far greater accuracy and precision by Messrs. Petit and Serres, under the designation of '*Fièvre Entéro-mésentérique.*' These observers pointed out that the lesions were limited to the lower portion of the ileum, and that hence the disease differed from ordinary enteritis. They were the first to regard it as specific. They expressed the opinion that the morbid appearances in the intestine resulted from the introduction of a poison into the system, and that they were of an eruptive nature, like the pustules of variola. Still, they believed that the abdominal lesions preceded, and were the cause of, the pyrexia, and that the extent of the former determined the severity of the latter. They also failed clearly to localize the disease in the solitary and agminated glands.[r]

After this, Cruveilhier,[s] Lerminier, and Andral[t] described the intestinal lesion as an internal exanthem, the ulcerations as preceded by '*pustules*,' and the larger patches as an '*anthrax de la* ' *membrane muqueuse.*' Andral, moreover, maintained that there was nothing to show that the disease commenced in the mucous follicles, and he classified Continued Fever under diseases of the abdomen.[u]

It was reserved for Bretonneau, of Tours, to prove that the disease was always localized in the solitary and agminated glands of the ileum. He also was the first to maintain that it depended on the action of a poison, which was communicable from the sick to the healthy. Although he considered the disease of the intestinal glands as inflammatory, and accordingly named the affection 'dothiénentérie' or 'dothiénentérite' (*δοθιὴν*, a tumour, and *ἔντερον*,

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[q] Broussais, 1816 and 1823.
[r] Petit and Serres, 1813, pp. 159, 165, and introd. pp. 20, 39.
[s] Cruveilhier, 1816. [t] Lerminier & Andral, 1823, i. 403. [u] Ib. 2nd ed. 1834.

intestine); he distinguished this inflammation from all other inflammations of the bowel; he showed that there was no correspondence between the severity of the febrile symptoms, and the extent of the intestinal lesion; and, like Petit and Serres, he insisted on the analogy of the latter to the cutaneous eruptions of the exanthemata. Bretonneau's views were made known in Paris in 1820, but were first published by his pupils Landini and Trousseau, in 1826, and by himself in 1829.[x] In 1829 was published the first edition of the elaborate and philosophic work of M. Louis.[y] Its appearance constituted an important epoch in the history of continued fevers, as the work furnished a standard of comparison with other fevers. Louis gave to the disease the unfortunate appellation of *Fièvre typhoïde*, which was adopted by Chomel in his Clinical Lectures, published in 1834,[z] and since then has come into general use. By the works of Louis and Chomel, it was shown that disease of the solitary and agminated glands of the ileum was always present in the fever of Paris; both authors, however, agreed that the severity of the fever did not correspond with the extent of the local disorder, and they described cases of *latent* typhoid, where the symptoms were extremely mild up to the date of fatal perforation. They also insisted on the necessity of not confounding typhoid fever with *gastro-entérite*.

All these French observers, however, regarded the contagious typhus of camps and armies, and of English writers, as identical with the disease under their own observation. Broussais remarked: 'En effet, puis que le mot typhus est synonyme du mot gastro-'entérite, chaque fois que l'on dira typhus des prisons, typhus des 'hôpitaux, typhus d'Amérique, typhus de Levant, ce sera, comme 'si l'on disait gastro-entérite des prisons, des hopitaux,' etc. Louis, Bretonneau, and Chomel were all inclined to regard the two affections as identical, although the two latter deplored the absence of careful *post-mortem* records of typhus cases, which, they thought, could alone decide the question. Chomel, after expressing doubts as to the contagious nature of 'typhoid fever,' remarked: '*Si des observations ultérieures démontraient*, dans le typhus, des 'lésions anatomiques semblables à celles que l'on rencontre dans la 'maladie typhoïde, l'identité de ces deux affections serait mise 'hors de doute, et la question de la contagion serait jugée.'

But, while French pathologists were thus maintaining that continued fever was always associated with disease of the intestinal

[x] Landini, 1826; Trousseau, 1826; Bretonneau, 1829.
[y] Louis, 1829. [z] Chomel, 1834.

and mesenteric glands, observers of fevers in Britain were making the discovery, that, in most fatal cases of fever, these parts were healthy. At the same time, observations, similar to those made in France, were not wanting in Great Britain and Ireland. In 1806, Dr. Thomas Sutton published the account of a '*remittent* '*fever*' among the troops at Deal, which was accompanied by great sickness and diarrhœa, while, after death, the bowels were found to be inflamed and gangrenous.[a] Willan and Bateman, in their Reports on the Diseases of London, between the years 1796 and 1816, make frequent mention of the same fever, as prevailing more particularly in autumn.[b] Many of the cases, bled so largely by Mills, at Dublin, in 1812, were evidently examples of the same fever.[c] To Dr. James Muir, we are indebted for an excellent history of a limited outbreak of pythogenic fever in the suburbs of Paisley, in 1811;[d] and to Mr. Henry Edmonstone, for an equally lucid account of an outbreak at Newcastle, in the autumn of 1817.[e] Mr. Edmonstone's account is particularly interesting, as it forms a striking contrast to the descriptions of typhus then prevailing in many other parts of the United Kingdom, and which afterwards visited Newcastle itself. The outbreak commenced in June, during extremely hot weather, following much rain, and lasted only six weeks. It was believed not to be contagious, and several members of a family were observed to be attacked simultaneously. Many of the first cases occurred in the higher ranks of life, and among servants in the best ventilated parts of the town, and it was scarcely known in those parts of the town where the infectious typhus was most common among the poor. Children and persons in the vigour of life were almost exclusively affected. Its duration was from 14 days to a month. Among the symptoms were vomiting, purging, melœna, epistaxis: cerebral symptoms were rare. Abercrombie, in 1820, recorded two cases of '*entero-mesen-* '*teric fever,*' in which the characteristic lesions of pythogenic fever were found after death, and stated that the so-called 'remit- 'tent fever' of infants was often symptomatic of intestinal disease.[f] In 1826, Dr. Hewett, of St. George's Hospital, published a number of cases, proving the frequent occurrence of 'follicular ulceration' of the bowels, in the idiopathic fever of London. Dr. Hewett's investigations have met with unmerited neglect. They were published almost simultaneously with those of Bretonneau; and, like his, they showed that the seat of the lesion was in the solitary and

[a] Sutton, 1806. [b] Willan, 1801, p. 25; Bateman, 1819, p. 145, etc.
[c] Mills, 1813. [d] Muir, 1811. [e] Edmonstone, 1818.
[f] Abercrombie, 1820.

agminated glands of the ileum. According to Dr. Hewett, the orifices of these glands became plugged up, the glands themselves distended with secretion, while the surrounding tissues became disorganized, partly by ulceration and partly by sloughing.[g] In 1827, Bright published his observations on fever in London, illustrated by excellent coloured drawings of the intestinal disease, which he spoke of as occurring occasionally.[h] In the same year, Dr. Alison stated that he had met with the intestinal affection described by French authors, at Edinburgh; but he maintained that it was not found after death from the ordinary typhus of that city; in 25 autopsies he had found Peyer's glands healthy.[i] In 1830, Dr. Tweedie[k] and Dr. Southwood Smith,[l] the two physicians of the London Fever Hospital, published the results of their experience. Both authors recorded a number of cases of fever, in which the intestines were found ulcerated, and the mesenteric glands enlarged after death, and other cases where these parts were healthy: both regarded the intestinal lesion, as merely one of many other complications of fever. A few years later (1834—7), Craigie confirmed Alison's observation, to the effect that, in the fever of Edinburgh, intestinal disease occasionally 'coexisted with 'the fever, and determined the fatal termination,' but that, in most cases, the intestines were healthy.[m] Lastly, although Irish observers ascertained that intestinal disease was exceptional in the fever of their own country; yet, in 1833, Dr. Cheyne described the symptoms and lesions of the fever of France, under the appellation of '*epidemic gastric fever*,' and stated that he had frequently observed it at Dublin;[n] and, in the following year, Mr. Poole adopted Cheyne's appellation, in his account of two outbreaks of the disease in different parts of Ireland.[o]

Thus, the French pathologists rarely failed to find the intestines diseased in fever: the English, on the contrary, in most cases found them healthy, and believing, either that the primary seat of fever was in the brain, or that fever was an idiopathic or essential affection, regarded the intestinal lesion as an accidental complication. In both countries, among the first effects of the increased study of morbid anatomy, was the neglect of the distinctions drawn in the previous century, between the slow nervous fever and the malignant fever of armies and jails.

Somewhat clearer views on the subject prevailed in Germany.

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[g] Hewett, 1826. [h] Bright, 1827. [i] Alison, 1827.
[k] Tweedie, 1830. [l] Smith, 1830.
[m] Craigie, 1834, 1836 and 1837. [n] Cheyne, 1833. [o] Poole, 1834.

In 1810, Hildenbrand distinguished between the *contagious typhus* and the *non-contagious nervous fever*;[p] and soon afterwards, many German writers regarded the *Typhus exanthematicus*, and the *Typhus abdominalis, Typhus gangliaris*, or *Nervenfieber*, as well-marked varieties.[q] The distinctions, however, which they laid down, were not sufficient to ensure any accuracy in diagnosis, still less to establish the non-identity of the diseases in question, while in 1844, Dr. Kuchler published a memoir to prove that they were identical.[r]

But the investigation of the question was soon to be renewed, and to be crowned with results, which even Erasmus Darwin could little have anticipated. A record of the successive steps, by which our present knowledge has been attained, is an important chapter in the history of Medicine.

Early in 1835, Dr. Peebles, who had observed the rubeoloid eruption in the contagious typhus of Italy, pointed it out to Dr. Perry, in the Glasgow Hospital. Dr. A. P. Stewart was present on the occasion, and from that date, the eruption, which seems to have been previously overlooked at Glasgow,[s] was noted in the majority of cases. In January, 1836, Dr. Perry published a paper, in which he correctly described many of the distinctions between typhus and enteric fever.[t] He referred to the complete absence of the 'typhus eruption' in 'dothinenteritis,' but did not state that the latter was characterized by an eruption of its own, although, four years later, Dr. Stewart remarked that Dr. Perry was the first, whom he had heard maintain the complete absence of the two eruptions.[u] The following extract, however, from Dr. Perry's memoir, shows that his ideas on the subject were far from being clear, and that he believed the existence of intestinal lesion to be not incompatible with true typhus.

'*Dothinenteritis* or enlargement of the mucous follicles of the 'smaller intestines, and enlargement and ulceration of the aggre-'gated glands of the lower third of the ileum, occurs *in combination* '*with contagious typhus*, and is to be met with in about six of those 'who die from typhus. It also exists as a disease *per se*.'

In the same year (1836), Dr. H. C. Lombard, of Geneva, who had previously had ample experience of pythogenic fever in Switzerland and France, visited various towns in England, Scotland, and Ireland. After watching some cases of fever in Glasgow and Dublin, which he had considered similar to the fever of the

[p] Hildenbrand, 1811, p. 15. [q] Reuss, 1814; Autenrieth, 1822; Stannius, 1835; Ebel and Grossheim, 1836; Schönlein, 1839.
[r] See Grisolle, *Path. Int.* 1852, i. 53. [s] See *antea*, p 46.
[t] Perry, 1836. [u] Stewart, 1840.

Continent, he was astonished to find no disease of Peyer's glands. After further investigations in Liverpool, Manchester, Birmingham, and London, he was the first to state that there were 'two distinct 'and separate fevers in Great Britain; one of them identical with 'the contagious typhus, the other a sporadic disease, identical with 'the typhoid fever, or *dothinenteritis* of the French.' He did not, however, determine the differences between the eruptions and the symptoms of the two fevers.[x] Almost at the same time, Messrs. Gerhard and Pennock, of Philadelphia, were arriving at the same conclusions, from observations of an epidemic of typhus, which prevailed in that city in the spring and summer of 1836. Both had previously studied enteric fever at Paris, and were familiar with it in their own country. They at once recognized the difference of the new disease, and, after a time, they were never deceived in their diagnosis. Their observations were published by Gerhard, in February and August, 1837. Gerhard maintained that the typhus of Philadelphia was identical with British typhus, and with the jail, camp, ship, petechial, or spotted fever, and that it was eminently contagious, while, on the other hand, enteric fever was rarely communicated. He showed that the lesions of Peyer's patches, and of the mesenteric glands, invariably present in the latter, were never found in the former, and remarked that English observers erred in regarding the intestinal disease as a mere complication of typhus. He insisted on the 'marked differ-'ence between the petechial eruption of typhus and the rose-'coloured spots of typhoid fever;' and he showed that a peculiar train of symptoms, very different from those of typhus, were associated with the intestinal affection, and that 'the distinctive 'characters of the two diseases were such as in practice could not 'allow them to be confounded.'[y] To Messrs. Gerhard and Pennock certainly belongs the credit of first clearly establishing the most important points of distinction between the two diseases. M. Valleix, of Paris, in a review published in January and February, 1839, thus alluded to Gerhard's observations: 'M. Gerhard établit 'd'abord un fait bien important, c'est qu'il peut exister, et qu'il 'existe en effet, concurrément dans le même pays, deux maladies, 'qu'on peut parfaitement diagnostiquer, et dans lesquelles, on peut 'prédire, pendant la vie du malade, les lésions qui seront trouvées 'après la mort: ce sont, la fièvre typhoïde, et le typhus propre-'ment dit.'[z]

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[x] Lombard, 1836. [y] Gerhard, 1837, xx. 289, 291, etc. [z] Valleix, 1839 (No. 1).

In 1837, the Académie de Médecine of Paris awarded prizes to the authors of two essays on the '*Analogies and Differences of 'Typhus and Typhoid Fever.*' These essays did not contain original observations bearing on the point; but referred chiefly to the previous records of the two diseases. One author (Gaultier de Claubry[a]) expressed his conviction that the two diseases were identical; the other (Montault[b]) arrived at the conclusion that, notwithstanding certain resemblances in their symptoms, they were really distinct. It may be remarked, that De Claubry, like some recent writers, although believing the two fevers to be identical, argued from the statements of previous observers, as if they had always employed the terms 'typhus' and 'typhoid' with strict accuracy. It was not surprising, then, that he maintained that intestinal lesions might exist in true typhus. It was De Claubry's memoir, however, that mostly influenced public opinion in France.

In 1838, Dr. Staberoh, of Berlin, after studying fever for four or five years at Vienna and Paris, and for six months in Britain, pointed out to the hospital physicians of Glasgow, the different eruptions met with in Continued Fevers, and remarked that these distinctions would facilitate the decision of the question of the specific difference of *typhus abdominalis* and *typhus exanthematicus.*[c]

In February, 1839, Dr. Shattuck, of Boston, U.S., came over from Paris, where he had already studied enteric fever, and watched some cases at the London Fever Hospital. He wrote an account of 13 cases, which he communicated to the Medical Society of Observation of Paris. About one-half of Dr. Shattuck's cases appear to have been typhus; the other half were enteric. Dr. Shattuck strongly insisted on the existence of two fevers in England, and pointed out with considerable minuteness the distinctions between them.[d] His paper formed the ground-work for a second review on fever, published by M. Valleix, in October, 1839, in which the conclusions were arrived at, that both typhus and *fièvre typhoïde* were to be met with in England, that the latter was the same as the fever of France, and that English practitioners erred in confounding them.[e]

In February, 1840, M. Rochoux published a memoir, in which he endeavoured to show that the 'dothiénentérite' of Bretonneau differed from typhus in its anatomical lesions, symptoms, and

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[a] De Claubry, 1838. [b] Montault, 1838. [c] Staberoh, 1838, p. 427.
[d] Shattuck, 1839. [e] Valleix, 1839 (No. 2).

causes. He insisted that nothing could be more unlike than the eruptions of the two fevers, and that while typhus was highly contagious, and generally believed to result from over-crowding, the contagious character of dothienenteritis was doubtful, and it was independent of over-crowding.[f]

On the 6th of the same month (February, 1840,) Dr. H. C. Barlow read a paper 'On the Distinction between Typhus Fever 'and Dothiénentérie,' before the Parisian Medical Society, which was published in abstract in *The Lancet* for February 29th. This paper has received less attention from subsequent writers than it deserves. Dr. Barlow maintained that typhus was an epidemic and highly contagious disease, and was usually most prevalent in winter; whereas, dothiénentérie was an endemic disease, but slightly, if at all, contagious, and always most prevalent in summer and autumn, along with other abdominal affections. Although typhoid symptoms were common to both fevers, as well as to other diseases, he showed that their clinical history and duration were entirely different. He carefully distinguished between the rose-coloured lenticular spots of the one disease, and the petechial eruption of the other, and he insisted that the lesions of dothiénentérie were never present in typhus. 'Surely,' he says, in conclusion, 'two diseases which differ in all these particulars 'cannot be identical.'[g]

Dr. A. P. Stewart (now physician to the Middlesex Hospital) studied fever in the Glasgow Fever Hospital, from the summer of 1836 to June, 1838, and afterwards at Paris. The results of his researches were communicated to the Parisian Medical Society on the 16th and 23rd of April, 1840, and were published in October of the same year. Dr. Stewart described, in a masterly manner, the leading distinctions between 'typhus' and 'typhoid' fevers, as regards their origin, proximate causes, course, symptoms, and anatomical lesions; and he supported his views by a statistical analysis of cases of both fevers. He pointed out more accurately than any previous observer, the differences of the eruptions; and he remarked that the characters of the two diseases, when taken collectively, were 'so marked as to defy misconception, and to 'enable the observer to form, with the utmost precision, the diag- 'nosis of the nature of the disease, and the lesions to be revealed 'by dissection.' He showed that, while there was overwhelming evidence to prove that the effluvia from living bodies, in close and unventilated localities, could generate the poison of typhus, 'typhoid

[f] Rochoux, 1840. [g] Barlow, 1840.

'fever' often appeared in country places, and in the best aired houses. The facts and arguments adduced in his memoir forced upon him the conviction, that the two fevers were 'totally 'different diseases.'[h] In November, 1840, a review of Dr. Stewart's memoir appeared in the '*Archives Générales de Méde-*'*cine*,' which the writer ended by remarking, that Dr. Stewart's observations demonstrated that in England there were two distinct diseases,—'typhus,' and 'typhoid fever.'

In consequence of the various researches now mentioned, Louis, in the second edition of his great work on 'Fièvre typhoïde,' published in 1841, admitted that, 'le *typhus fever* des Anglais est 'nécessairement une maladie très différente de celle qui nous 'occupe'; and he added, that although difficulties in diagnosis might occasionally arise, such difficulties were encountered in the diagnosis of the best-known diseases, and in no way detracted from the specific non-identity of the two fevers in question.[i] Bartlett, also, in the first edition of his work on American Fevers, treated them as distinct diseases.[k] In 1846, Dr. Ritchie, of Glasgow, accurately described the various circumstances in which the two fevers agreed and differed;[l] and since 1846, the cases of each fever admitted into the Glasgow Royal Infirmary have been carefully distinguished. In the following year (1847) Dr. H. Guéneau de Mussy came over from Paris to Dublin, and after studying typhus, which was then so prevalent in that city, was convinced of its specific distinctness from the fever of Paris. On his return to Paris, his arguments induced M. Grisolle to adopt the same view.[m] Dr. De Mussy observed one case at Dublin where a patient died of typhus contracted during convalescence from enteric fever; the cicatrices of the intestinal ulcers were discovered after death.

The doctrine of non-identity, however, did not remain unopposed. In a careful review of the subject, published in July and October, 1841, the writer,[n] with all the evidence before him, regarded the two fevers as varieties, but not distinct species. Dr. Davidson, in the Thackeray Prize Essay on Fever (1840), came to the same conclusion.[o] In June, 1845, De Claubry reiterated to the French

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[h] STEWART, 1840 and 1858. [i] LOUIS, 1841, ii. 318, 324. [k] BARTLETT, 1842.
[l] RITCHIE, 1846. [m] GRISOLLE, *Path. Int.*, 1852, i. 55.
[n] See *Bibliogr.*, 1841, *Review*.
[o] DAVIDSON, 1841. It may be mentioned that Dr. William Budd, now of Bristol, was another candidate for the prize given by the late Dr. Thackeray, for the best essay on the 'Causes and Mode of Propagation of the Continued Fevers of Great Britain.' His essay was never published, but had three principal objects. 'The first was to prove, by various evidence, that the typhoid

Academy of Medicine, his belief in identity, although in the subsequent discussion, he was strongly opposed by Rochoux. Dr. Waters, also, in his inaugural Prize Thesis, presented to the Medical Faculty of the University of Edinburgh, in 1847, stated that the conclusion was inevitable, that the two fevers were identical.[p] Indeed, notwithstanding the decided opinions expressed by the several observers above-mentioned, it was the general impression, both in England and France, that the evidence adduced was insufficient to establish the specific non-identity of the two fevers, and the opposite doctrine continued to be taught in all medical schools.

Much of the remaining doubt, however, was removed by the admirable researches of Dr. W. Jenner, published between 1849 and 1851. Dr. Jenner confirmed and amplified the distinctions between the symptoms of the two diseases previously drawn by Gerhard, Stewart, and others, and did much to facilitate their diagnosis. He supported his statements by carefully-recorded cases, and by an elaborate analysis of the symptoms and *post-mortem* appearances of numerous cases of both fevers observed by him at the London Fever Hospital. But the most important part of his investigations bearing on the question at issue, was that which demonstrated the dependence of the two fevers on distinct causes. By an analysis of all the cases admitted into the London Fever Hospital, for more than two years, he showed that the two fevers did not prevail together, and that the one did not communicate the other. He also adduced cases to prove that an attack of the one fever protected from subsequent attacks of itself; but not of the other. Dr. Jenner maintained, that typhus, and the so-called 'typhoid [fever,' were as distinct as any two of the exanthemata.[q]

During the last ten years, many physicians enjoying independent spheres of observation, have arrived at the same conclusions as Gerhard, Stewart, and Jenner. Among our own countrymen may be mentioned Dr. Peacock,[r] of St. Thomas's Hospital; Dr. Wilks,[s] of Guy's Hospital; Dr. Watson[t] and Dr. Tweedie;[u] Dr.

'fever and the maculated typhus—the fever with intestinal affection and the 'fever without such affection—are not varieties merely of one disease, but 'two diseases of essentially different species. The second, that both species 'are essentially contagious. The third, that there is no valid evidence to show 'that the specific poison, from which either respectively springs, is ever bred 'elsewhere than in the living and already affected body.' (Budd, 1859, p. 5).

[p] Waters, 1847 (not published). [q] Jenner, 1849, 1850, and 1853.

[r] Peacock, 1856 and 1862. [s] Wilks, 1855 and 1856.

[t] Watson, *Lectures on Physic*, 4th ed. 1857, vol. ii. [u] Tweedie, 1860.

W. T. Gairdner,[v] of Edinburgh; Dr. Anderson,[x] of Glasgow; and Dr. Lyons,[y] of Dublin. In an essay, presented to the Medical and Chirurgical Society of London, in 1858, I endeavoured to show that the two fevers were very different in their mode of origin.[z] In America, the non-identity of the two fevers has been advocated by Bartlett,[a] Austin Flint,[b] and Wood,[c] and is generally recognized. Many continental physicians also, who have lately had an opportunity of studying typhus, have expressed their conviction of its distinctness from the enteric fever, with which they had been more familiar previously. In 1854, Forget communicated to the French Academy of Sciences, the report of an epidemic of typhus in the jail of Strasbourg. Although in his work on 'Entérite 'Folliculeuse,' published in 1841, he had expressed his belief that the diseases were identical, his first experience of true typhus led him to an opposite conclusion, and in the memoir referred to, he uses the following words:—'J' expose une série d' observations 'avec autopsie, qui démontrent l' absence de l' entérite folliculeuse 'dans le typhus. Comme corollaire des faits précédents, j'établis 'un parallèle entre les deux maladies, d'où resulte qu'elles 'diffèrent, non seulement par les caractères anatomiques, mais 'encore par les causes, les symptômes, la marche, la durée, et le 'traitement.'[d] The French physicians, who met with typhus during the Crimean war, adopted the doctrine of non-identity, almost without exception. Two of them may be referred to, by way of illustration. In 1856, Godélier communicated to the French Academy an excellent report of 63 cases of typhus observed at the Hospital of Val-de-Grâce. He maintained that British typhus was identical with the typhus of prisons and armies, but differed entirely from the *fièvre typhoïde*, in its mode of origin, symptoms, and anatomical lesions. 'Le typhus, et le *typhus* '*fever* sont identiques; ils diffèrent spécifiquement de la *fièvre* '*typhoïde*.'[e] Jacquot summed up the evidence on the question as follows:—'En un mot, chaque espèce, typhus et fièvre typhoïde, 'présente tous les degrés d' intensité, sans cesser de garder son 'individualité, ses caractères, sa marche, ses symptômes, ses 'lésions.'[f] Again, M. Barrallier, in an important work on a recent epidemic of typhus at Toulon, enters minutely into the question of its distinctness from the ordinary fever of France, and remarks:—'Elles sont séparées l'une de l'autre par leurs causes,

[v] W. T. GAIRDNER, 1860 and 1862. [x] ANDERSON, 1861.
[y] LYONS, 1861. [z] MURCHISON, 1858. [a] BARTLETT, 1856.
[b] FLINT, 1852. [c] WOOD, *Treatise on the Practice of Medicine*, 4th ed. 1855.
[d] FORGET, 1854. [e] GODÉLIER, 1856, p. 896. [f] JACQUOT, 1858, p. 307.

'leurs symptômes, leur marche, leur durée, leurs caractères ana-
'tomiques; elles appartiennent réellement à la même classe de
'maladies, les fièvres essentielles spécifiques, mais elles con-
'stituent des genres à part, comme la rougeole et la scarlatine
'dans le groupe des fièvres éruptives.'[g]

Many German physicians, among whom may be mentioned Griesinger,[h] have adopted the same view.

The specific distinctness of the two diseases, is now, in fact, generally recognized in every part of the world. It is true that some excellent observers still adhere to the doctrine of identity,[i] and maintain that it is impossible to distinguish the symptoms or lesions of the two fevers, and that indeed the dothinenteritis of Bretonneau is merely an accidental complication of typhus. Looking at the past history of medicine, it would be surprising were it otherwise. The arguments on both sides of the question will be discussed in a subsequent chapter.

Section IV.—Geographical Distribution.

Pythogenic fever has been known to occur in almost every part of the world.

It is endemic in the British Isles, but is apparently most common in England, more common in Ireland than in Scotland, and more common in the west coast of Scotland than in the east coast. Of 2431 cases of pythogenic fever admitted into the London Fever Hospital during fourteen years, (1848-1861), the birth-place was noted in 2005, as follows:—

Table XXVIII.

Natives of London,	1072,	or 53·47	per cent.
,, of rest of England,	741,	,, 36·95	,,
,, of Scotland,	13,	,, .64	,,
,, of Ireland,	162,	,, 8·08	,,
Foreigners,	17,	,, ·84	,,
Total,	2005	99·98	,,

Taking the census of 1851 (see page 57) as a basis of information concerning the birth-place of all the inhabitants of London, it follows that during the period above mentioned there were admitted into the London Fever Hospital

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[g] Barrallier, 1861, p. 129. [h] Griesinger, 1853.

[i] Christison, 1858; Stokes, 1854; Kennedy, 1860 and 1862; J. Bell, 1860; Huss, 1855; Yates, 1857; Chambers, 1858; Barclay, *On Med. Diagnosis*, 1859; Barlow, *Man. of Pract. of Med.* 1856; J. H. Bennett, *Clinical Lectures*, 1859.

1 in every 670 of the Irish inhabitants.
1 „ 1208 of the English inhabitants.
1 „ 1906 of Foreigners.
1 „ 2338 of the Scotch inhabitants.

The contrast here presented with typhus and relapsing fever will be seen by referring to pages 57 and 300. Of the Irish inhabitants of London, 1 in 310 was admitted with typhus, and 1 in 386 with relapsing fever, but only 1 in 670 with pythogenic fever. Of the English inhabitants, 1 in 1208 was admitted with pythogenic fever, but only 1 in 16,465 with relapsing fever. There was no evidence that pythogenic fever was ever imported from Ireland. Of the 162 Irish patients, all but 9 had resided in London more than three months, and in only two or three of the 9 cases does it appear that the patients had come direct from Ireland.

Medical literature abounds with records showing pythogenic fever to be endemic in France, Germany, Russia, Spain, Italy, and Turkey. Many of these records are referred to in this work; others are alluded to in the elaborate collection of Hirsch.[k] Its occurrence in Norway and Sweden has been demonstrated by Huss,[l] Conradi,[m] etc., and in Iceland by Schleisner[n] and Hjaltelin.

Unlike typhus, pythogenic fever is often met with in the tropics, where it has probably been often mistaken for remittent fever. In India, it is now known to be far from uncommon. Twining long ago pointed out, that a fever often prevailed in Bengal, which proved fatal under typhoid symptoms, and in which the small intestines were found ulcerated after death.[o] Similar observations were made in Madras, by Mouat and Shanks,[p] while the recent researches of Scriven,[q] Ewart,[r] Edward Goodeve,[s] Cornish,[t] and Ranking,[u] leave no doubt on the matter. These gentlemen have recorded numerous cases of fever occurring in various parts of the Bengal and Madras Presidencies, and in Burmah, which, in their symptoms, (including the eruption) and *post-mortem* appearances, agreed, in every respect, with the so-called 'typhoid fever' of French and English writers. Dr. Morehead, also, appears to have met with it in Bombay.[x] Heymann has frequently observed it in Sumatra and Java,[y] and it has also been shown to prevail in Syria.[z]

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[k] HIRSCH, 1859. [l] HUSS, 1855. [m] HIRSCH, 1859, p. 158. [n] SCHLEISNER, 1850.
[o] TWINING, *Diseases of Bengal*, 1832, p. 13. [p] HIRSCH, 1859, p. 161.
[q] SCRIVEN, 1854 and 1857. [r] EWART, 1856. [s] GOODEVE, 1859.
[t] CORNISH, 1862. [u] RANKING, 1862. [x] MOREHEAD, *Researches on Disease in India*, 2nd ed. 1860, p. 160. [y] SCHMIDT'S *Jahrb. Bd.* lii. 96. [z] HIRSCH, 1859, p. 160.

In Africa it is not wanting. Haspel,[a] Cambay,[b] and other French writers have observed it in Algeria. Griesinger[c] mentions it as occurring in Egypt; and Oelsner, in the Isle of Bourbon.[d] It is probably not uncommon on the West Coast of Africa. M'William, in his account of the Niger Expedition,[e] records the *post mortem* appearances of several cases of fever, as follows:— 'The jejunum was free from disease, and likewise the ileum, until 'within three feet of its lower end, where were observed softening 'of the mucous lining generally, and livid spots. A series of 'small ulcerations were seen in 4 cases. In one the membrane was 'thickened and rough, and the ulceration had nearly perforated the 'bowel. The agminated glands of Peyer were distinct, and en- 'larged in 3 cases. The morbid appearances observed in the 'intestines are very like those so often found in fatal cases of the 'typhoid fever of this country.' Again, in the Museum of Fort Pitt, there is a drawing showing the condition of the intestines, in a case which proved fatal at Sierra Leone, and which was believed to be yellow fever, but which was probably pythogenic fever complicated with jaundice.[f]

In North America, pythogenic fever is endemic from Greenland to the Gulf of Mexico. The writings of Gerhard,[g] Bartlett,[h] Flint,[i] Jackson,[k] and Wood,[l] are often referred to in this work, and many other references have been collected by Hirsch. Martinez del Rio,[m] Jecker,[m] Newton,[n] Stricker,[n] and Gibbs,[n] have described a fever as prevailing in Mexico, which presented all the symptoms and anatomical lesions of pythogenic fever; while Lidel[o] and Praslow,[o] have reported its occurrence in Central America and California. According to Tchudi, it is extremely common in Brazil and Peru.[o]

Lastly, pythogenic fever has been observed in New Zealand and Van Dieman's Land, by Power[p] and Milligan.[q]

Section V.—Etiology.

A.—Predisposing Causes.

1. *Sex.*

Pythogenic fever appears to attack one sex as readily as the other. Of 2,432 cases admitted into the London Fever Hospital during fourteen years (1848-61), 1,211 were males, and 1,221

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[a] Haspel, 1850. [b] Cambay, 1854. [c] Griesinger, 1853. [d] Hirsch, 1859, p. 162.
[e] London, 1843, p. 144. [f] Jenner, 1853, p. 312. [g] Gerhard, 1837.
[h] Bartlett, 1842 and 1856. [i] Flint, 1852. [k] Jackson, 1838.
[l] Wood, *Treat. on Pract. of Med.* 4th ed. 1855.
[m] Louis, 1841, vol. i. pref. p. 17. [n] Hirsch, 1859, p. 164. [o] Ibid.
[p] *Dublin Quarterly Journ.*, 1843, xxiii. 91. [q] Hirsch, 1859, p. 165.

were females; or the females only exceeded the males by 10. Of 2,312 cases collected by Bartlett from several American sources, 1,179 were males, and 1,163 females; or the males exceeded the females by 16.[r] Of 487 cases admitted into the Glasgow Infirmary from 1857 to 1861, 270 were males, and 217 females.[s] Of 138 cases observed by Louis, at Paris, only 32 were females; but the excess of males in this case was accounted for by the circumstance that a larger number of males were strangers in Paris, and could not be treated at their own homes.[t]

According to West,[u] pythogenic fever occurs more than twice as often in boys as in girls, and statistical data, as far as they go, corroborate the statement. Thus—

	Boys.	Girls.	Total.
Barthez and Rilliet[x]	80	31	111
Taupin[y]	86	35	121
London Fever Hospital, under 10 years, see Table XXIX.	72	35	107
	238	101	339

It may be doubted, however, whether this preponderance of males be not due to accidental circumstances (see page 61). Of 98 cases reported by Friedleben, 46 were boys, and 52 girls.[z]

2. *Age.*

The predisposition to pythogenic fever is much influenced by age, the disease being chiefly met with in youth and adolescence.

The mean age of 1,772 cases admitted into the London Fever Hospital during ten years, was 21·25; that for males being 21·45, and for females, 21·06. The mean age of 3,456 cases of typhus was ascertained to be 29·33. Table XXIX. shows the number admitted in each quinquennial period of life.

From this Table it appears that more than one half (52 per cent.) of the cases were between fifteen and twenty-five years of age, and one-fifth were under fifteen.[a] Less than one-seventh were above thirty, and only 1 in 68 exceeded fifty. The entire population of England and Wales in 1851, being 13,771,056

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[r] BARTLETT, 1856, p. 109. All but 98 of Bartlett's cases were fatal cases.
[s] *Hospital Reports.* [t] LOUIS, 1841, ii. 354.
[u] *Diseases of Children*, 3rd ed. 1854, p. 561.
[x] BARTHEZ and RILLIET, 1853, ii. 714. [y] TAUPIN, 1839.
[z] *Brit. & For. Med. Chir. Rev.*, July, 1858, p. 161.

[a] The proportion of pythogenic cases in early life would be still greater, were it not that many children labouring under this disease are treated at dispensaries, and at their own homes, as cases of 'Infantile Remittent Fever,' and that comparatively few young children are admitted into the Fever Hospital.

TABLE XXIX.

Age.	No. of Cases.			Per centage at each period.
	M.	F.	M. & F.	
Under 5 years	2	2	4	·23
From 5 to 10 years ...	70	33	103	5·81
,, 10 to 15 ,, ..	121	129	250	14·11
,, 15 to 20 ,, ...	237	282	519	29·29
,, 20 to 25 ,, ...	191	213	404	22·8
,, 25 to 30 ,, ...	123	117	240	13·54
,, 30 to 35 ,, ..	50	50	100	5·64
,, 35 to 40 ,, ...	31	29	60	3·39
,, 40 to 45 ,, ...	28	18	46	2·59
,, 45 to 50 ,, . .	9	11	20	1·13
,, 50 to 55 ,, ...	5	3	8	·45
,, 55 to 60 ,, ...	6	3	9	·51
,, 60 to 65 ,, ...	3	4	7	·39
,, 65 to 70 ,, ...	1	—	1	·05
,, 75 to 80 ,, ...	1(?)	—	1(?)	·05
Age doubtful	27	21	48	—
Total, omitting doubtful cases	878	894	1772	99·98

persons under thirty years of age, and 4,338,354 above thirty, it follows that persons under thirty are twice as liable to pythogenic fever as persons over thirty. The difference in this respect from typhus, is remarkable (See page 62). The contrast between the ages of the typhus and pythogenic cases admitted into the London Fever Hospital, is also strikingly brought out by the following comparison—

	Per cent. of Typhus cases.	Per cent. of Pythogenic cases.
Under 10 years there were . .	5·78	6·04
,, 15 ,, ,, . .	16·3	20·14
From 15 to 25 years ,, . .	30·12	52·08
25 years and upwards . .	53·58	27·76
30 ,, ,, . .	43·66	14·22
40 ,, ,, . .	26·47	5·19
50 ,, ,, . .	11·92	1·46
60 ,, ,, . .	4·68	·5

The increase in the number of cases between forty and forty-five years of age, observed in typhus and relapsing fever (see pages 63 and 304), did not occur in pythogenic fever.

There was little difference between the ages of males and females. In some years, the mean age of the males was greater; in others,

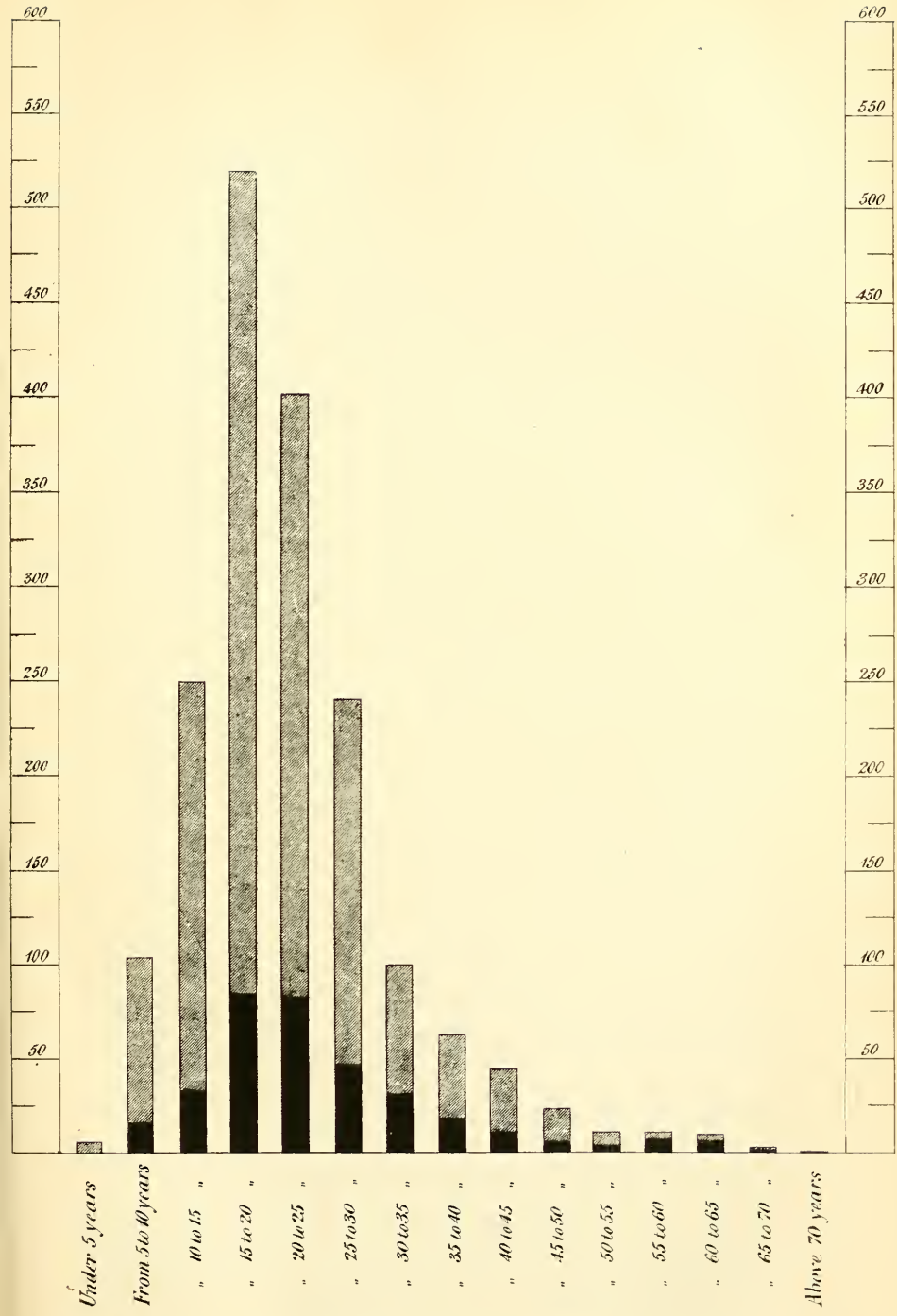

Diagram VI, shows the ages of 1772 cases of Pythogenic Fever, admitted into the London Fever Hospital, with the number of deaths ■ *at each age. (Compare with Diagr. II.)*

that of the females; and for the whole ten years, the mean age of the two sexes was, as above stated, almost equal. Of 1620 cases under thirty, the females exceeded the males by 32; and of 252 cases over thirty, the males exceeded the females by 16; this result, as far as it goes, is contrary to what was noted in typhus and relapsing fever.

The fact that pythogenic fever is mainly a disease of young persons, has been confirmed by every observer.[b] For reasons already stated, cases are more common in infancy and childhood than the returns of the London Fever Hospital might lead one to believe. Of 7,348 cases reported to the French Academy, from different parts of France, Gaultier De Claubry ascertained that 2,282, or 31 per cent. had not attained fifteen years of age.[c] The youngest cases observed at the London Fever Hospital in the period above-mentioned, were 3 children aged four. Many years ago, M. Rufz endeavoured to show that the disease did not occur under four years of age,[d] and West states that it is rare under five years.[e] There are many cases on record, however, of its occurrence in the third and fourth years of life;[f] and indeed, although they are rare, there are not wanting authenticated examples in infants under two years of age. Cases of its occurrence in the first year of life have been recorded by Abercrombie,[g] Rilliet,[h] Friedrich,[i] Hennig, and Wunderlich.[k] M. Charcellay, a colleague of Bretonneau's in the hospital at Tours, has published two cases of the disease in newly-born children. One died on the eighth, and the other on the fifteenth day after birth; in the former, it was concluded both from the symptoms and *post-mortem* appearances, that the disease must have been contracted in the mother's womb, although the mother had not the fever, either during pregnancy, or after delivery.[l] About the same time also, Manzini communicated to the Académie des Sciences, the account of a dissection of a seven months' fœtus, which died within half an hour after birth, and in which many of Peyer's patches presented appearances similar to those of dothinenteritis; no mention is made of the mother having the fever.'[m]

On the other hand, youth is not necessary for the development

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[b] Louis, 1841, ii. 353: Chomel, 1834; Jenner, 1850, xxii. 457; Bartlett 1856, p. 107; Davenne, 1854. [c] Gaultier de Claubry, 1849, xiv. 29.
[d] Rufz, 1840. [e] West, *Dis. of Child.* 1854, p. 561.
[f] Rilliet and Barthez, 1840 and 1853; Taupin, 1839.
[g] Abercrombie, 1820. [h] Rilliet, 1853, ii. 713-4. [i] Friedrich, 1856.
[k] *Brit. & For. Med. Chir. Rev.* July, 1858, p. 161; see also *Gazette Méd.* viii. 717, ix. 781. [l] Charcellay, 1840. [m] Manzini, 1841.

of pythogenic fever, as Louis was inclined to think.[n] Although most observers have noted its rarity above fifty years of age, 26 of 1772 cases at the London Fever Hospital, or 1½ per cent., exceeded that age, a proportion which is much larger than at first appears, when it is remembered that less than one-seventh of the entire population of England and Wales is constituted by persons above fifty, and that many who survive that age have acquired an immunity from the disease by a previous attack. Nine cases were noted at the Fever Hospital above sixty; one of them, a man, had rose spots, and called himself seventy-six, but did not look more than sixty. Lombard,[o] Gendron,[p] and Reeves[q] mention 17 cases where the patient's age exceeded fifty; and Jacquez reports several cases where the age exceeded sixty, and one where it was more than seventy.[r] Trousseau records the case of a woman aged 64, in whose body the characteristic abdominal lesions were found after death.[s] These lesions have likewise been found, by Wilks, in a woman aged 70;[t] by Lombard, in a woman aged 72;[u] and, by M. D'Arcy, in a woman aged 86.[x]

3. *Mode of Prevalence.*

Pythogenic fever differs from typhus and relapsing fever, in being essentially an endemic disease. It is, in fact, the endemic fever of England, as it is of France and America. The following table shows the number of cases admitted into the London Fever Hospital, and Glasgow Infirmary, during the fourteen years that the disease has been distinguished from typhus:—

TABLE XXX.

Years.	Lond. Fever Hospital.	Glasgow Roy. Infirmary.	Years.	Lond. Fever Hospital.	Glasgow Roy. Infirmary.
			Brot. forward.	1240	315
1847	?	127	1855	217	145
1848	152	7	1856	149	163
1849	138	?	1857	214	157
1850	137	?	1858	180	117
1851	234	44	1859	176	87
1852	140	134	1860	94	91
1853	211	45	1861	161	36
1854	228	92	1862 To July 31	93	—
Total.	1240	315	Total.	2524	1111

From this table, it is obvious that the number of cases has varied little from year to year. The average number for the entire

[n] LOUIS, 1841, ii. 353. [o] LOMBARD et FAUCONNET, 1843, p. 591.
[p] GENDRON, 1829. [q] REEVES, 1859. [r] JACQUEZ, 1845.
[s] TROUSSEAU, 1859. [t] *Path. Soc. Trans.* vol. xiii. p. 68.
[u] LOMBARD et FAUCONNET, 1843, p. 592. [x] GAULTIER DE CLAUBRY, 1849, p. 30.

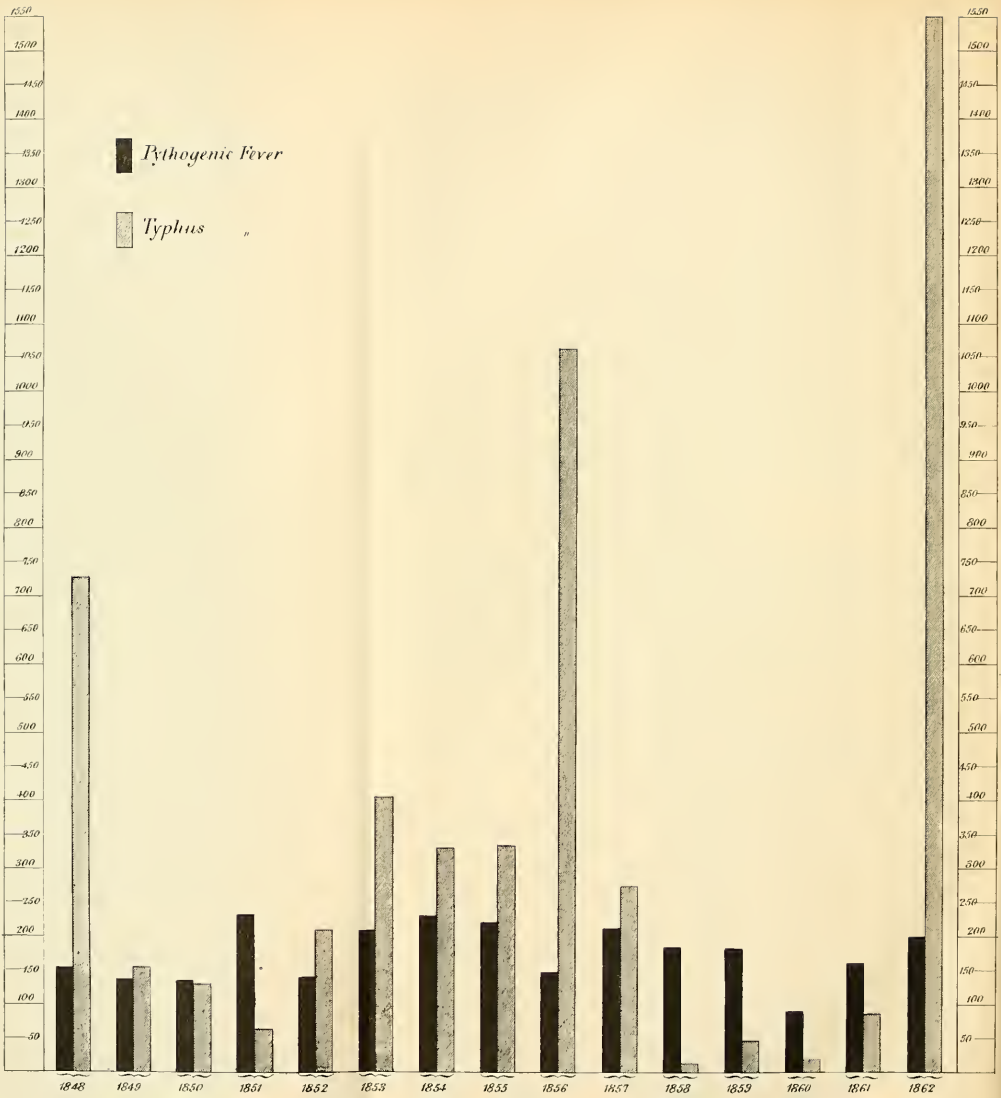

Diagram VII, shows the number of admissions into the London Fever Hospital, during fifteen years, of Pythogenic Fever and Typhus.

H. Hurst, Lith.

fourteen years being 174, the largest was 234, and the smallest 94; or eliminating the year 1860, which was exceptional for reasons hereafter mentioned, the average was 179; the largest number, 234; and the smallest, 137. Its mode of prevalence, then, presents a marked contrast to that of typhus, as may be seen by referring to Table II. (page 51), and to Diagrams I. and VII. Moreover, the extent of its prevalence is quite independent of that of typhus. Thus, in 1856, when 1,062 cases of typhus were admitted into the London Fever Hospital, the number of pythogenic cases did not exceed 149; but, in 1858, when the typhus cases had dwindled down to 15, the pythogenic cases did not decrease in like manner, neither did they increase to take the place of typhus, as has been stated. In fact, the admissions of pythogenic fever for the year 1858, corresponded exactly to the average of the ten preceding years, the former being 180, and the average 182. Again, there has been no increase or diminution of pythogenic fever during the present epidemic of typhus in London. During the first six months of the present year (1862), while no fewer than 1,107 cases of typhus were admitted into the Fever Hospital, the number of pythogenic cases amounted to 74; but during the first six months of 1858, when the typhus cases did not exceed 13, the pythogenic cases were 73, and during the first six months of 1851, when the typhus cases were only 36, the pythogenic cases were 81. Lastly, between April 20th and May 21st, 1862, 210 cases of typhus were admitted into the Fever Hospital, and not a single case of pythogenic fever, while, between the same dates of the preceding year (1861), only 1 case of pythogenic fever was admitted, and the typhus cases did not exceed 10.

In Glasgow, where the only other accurate returns have been kept, pythogenic fever appears to be also endemic, and to be independent of typhus in the extent of its prevalence. Thus, during the eleven years (1851—61), the annual admissions into the Royal Infirmary averaged 101, never exceeded 163, and were never less than 36, although the annual admissions for typhus varied from 175 to 1,551. Again, in 1858, there were 117 cases of pythogenic fever to 175 of typhus, whereas, in 1847, there were only 127 cases of pythogenic fever to 2,399 cases of typhus and 2,333 cases of relapsing fever.

So, also, in Edinburgh and its neighbourhood, pythogenic fever is, at least, as common now, when typhus is almost unknown, as it has been during some of the greatest epidemics of typhus. When on a visit to Edinburgh, in May, 1858, I ascertained that there were several cases of pythogenic fever in the Royal Infirmary, but not one case of typhus. Again, I am informed by my friend,

Dr. J. W. Begbie, that, from February to the end of July of the present year (1862), 20 cases of pythogenic fever were admitted, but only 3 of typhus. From all accounts, pythogenic fever has been somewhat more common in Edinburgh during the last few years than it was formerly; but this does not imply that it has taken the place of typhus. In the first place, it may be doubted if it was such a rare disease, in Edinburgh, and in its neighbourhood, in former years, as has been imagined. In 1827, Dr. Alison stated that he had frequently seen cases in children presenting all the symptoms and *post-mortem* appearances of the fever described by French writers.[y] Christison observed the same fever in 1829,[z] and a few years later, several cases came under the care of Dr. Craigie.[a] Some years ago, my friend, Dr. Haldane, placed at my disposal the *Post-mortem* Registers of the Royal Infirmary, which have been kept with great accuracy since 1832, and I found that in the first six years, 15 cases had been *dissected*, about which there could be little doubt. Thus :—

In 1833, there were	3 cases.	In 1836, there were	3 cases.
„ 1834, „	2 „	„ 1837, „	2 „
„ 1835, „	2 „	„ 1838, „	3 „

Of 132 cases of fever dissected by Dr. John Reid, between 1838 and the end of 1841, ulceration of Peyer's patches was present in 8; and only one-fourth of the fatal cases were examined.[b] In 1842, ulceration of Peyer's patches was found in 3 out of 29 cases; and in 85 fatal cases of fever, the intestines were not examined.[c] Between November 1st, 1846, and June, 1847, 19 cases of fever, with ulceration of Peyer's patches were dissected in the Royal Infirmary.[d] Since 1854, the cases of pythogenic fever dissected in the Edinburgh Infirmary, have been as follows :—

1854 . . .	5 cases.	1858 . . .	1 case.
1855 . . .	2 „	1859 . . .	2 „
1856 . . .	1 „	1860 . . .	1 „
1857 . . .	8 „	1861 . . .	6 „

These numbers do not indicate a very decided increase, especially when it is remembered that the greater attention devoted to the subject of late years has probably secured a larger number of autopsies in fatal cases. Moreover, it is probable, that before pythogenic fever was distinguished from typhus, it was often overlooked or confounded with local diseases of the intestines.

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[y] Alison, 1827. [z] Christison, 1858, p. 558.
[a] Craigie, 1834, 1836, and 1837. [b] Reid, 1840 and 1842.
[c] Peacock, 1843. [d] Bennett, 1847; Waters, 1847.

The late Dr. M'Ghie informed me that before the disease was recognized in Glasgow, many cases were recorded in the hospital books, under the title of *Muco-enteritis*, which were evidently examples of pythogenic fever, and the same remark probably applies to many of the cases described by Cullen and his contemporaries as *enteritis erythematica*. At the same time, there is reason to believe that a larger number of cases have originated in the *town* of Edinburgh of late years, than formerly. From the evidence of John Reid, Peacock, and Robertson,[e] it appears that, during the five years, 1838-42, and the three years preceding 1847, not one case of fever with intestinal lesion was dissected in the infirmary, in which the patient had contracted his illness in the town; but now Dr. W. T. Gairdner tells us, that the town enjoys no such immunity, and that a large proportion of the cases are indigenous.[f] Although this increase is probably, in a great measure, due to the disease being better known, it is doubtful if such an explanation is sufficient, and if it be not, it would be interesting to enquire, how far the increase has been connected with the introduction of new sanitary arrangements, with the substitution for the scavenger and nightman, of drains opening into the interior of the houses, but with a water-supply insufficient to prevent the escape of sewer-gases.[g] At all events, I venture to predict, that the prevalence of pythogenic fever will be neither increased nor diminished by an outbreak of typhus.

Pythogenic fever has formed no part of the great fever-epidemics which have devastated Britain, although, of course, cases are met with during these epidemics, just as we meet with cases of measles and small-pox.[h] But although essentially an endemic disease, it may become epidemic even in localities where, for years before, it has been unknown. These epidemics, however, are distinguished from epidemics of typhus, in being local and circumscribed. Sometimes they are confined to a single house or village. Many illustrations of such epidemics will be subsequently adduced; others will be found in the works of John Reid,[i] Stewart,[j] and Bartlett;[k] and particularly in the reports on epidemics presented to the French Academy.[l] Owing to the circumscribed character of its epidemics, pythogenic fever has often been named from the localities in which it occurs. Thus we read of the 'Croydon

[e] Robertson, 1848. [f] Gairdner, 1862 (No. 2), p. 170.
[g] See Gairdner, 1862 (No. 1), p. 255.
[h] See the accounts of the epidemics of 1826 and 1847, at pages 44 and 50.
[i] Reid, 1842. [j] Stewart, 1840 and 1858, p. 275.
[k] Bartlett, 1856, pp. 99, 106.
[l] See *Bibliography*, 1833, 1849, and 1850, and particularly 1849, p. 54.

Fever,' the 'Westminster Fever,' the 'Cowbridge Fever,' and the 'Windsor Fever.'

4. *Months and Seasons.*

Unlike typhus, pythogenic fever varies greatly in its prevalence, according to the months and seasons of the year. The monthly admissions during 14½ years into the London Fever Hospital are given in Table XXXI. Diagrams VIII. and IX. also show the admissions in the quarters and seasons of each year.

It is obvious, from Table XXXI. that by far the largest numbers have been admitted during the autumn months, September, October, August, and November, in the order here given, and the smallest in April, May, February, and March. In the two months, September and October, 28 per cent. of the entire number were admitted; but in April and May only 7 per cent. Moreover, this great increase of pythogenic fever in the autumn months was observed in each of the fourteen years, with one remarkable exception (1860) hereafter alluded to; and although the different continued fevers have only been registered at the Fever Hospital for fourteen years, I find, on referring to the printed reports for at least twenty years before, that ulceration of the bowels was always noted as most common during autumn. The contrast between pythogenic fever and typhus, in this respect, will be apparent on comparing Table XXXI. with Table VI. (page 65), and Diagrams VIII. and IX., with Diagrams III. and IV.

It is also worth noticing that the increased prevalence of pythogenic fever in autumn does not subside immediately on the advent of winter. In fact, in the winter months, (November, December, and January), the cases are much more numerous than in the summer months. The disease, which is at its maximum at the middle and end of autumn, continues to decrease until April, when it is at its minimum, and then progressively increases through the summer and autumn months. It would seem that the cause of the disease is only exaggerated or called into action by the *protracted* heat of summer and autumn, and that it requires the *protracted* cold of winter and spring to impair its activity or to destroy it.

The increased prevalence of pythogenic fever in autumn is not limited to the Fever Hospital, or to London. Numerous inquiries have convinced me that the same rule holds good at the other Metropolitan Hospitals. Of 131 cases treated by the late Dr. Todd, in King's College Hospital, during a period of twenty years, I ascertained that 21 were admitted in spring;

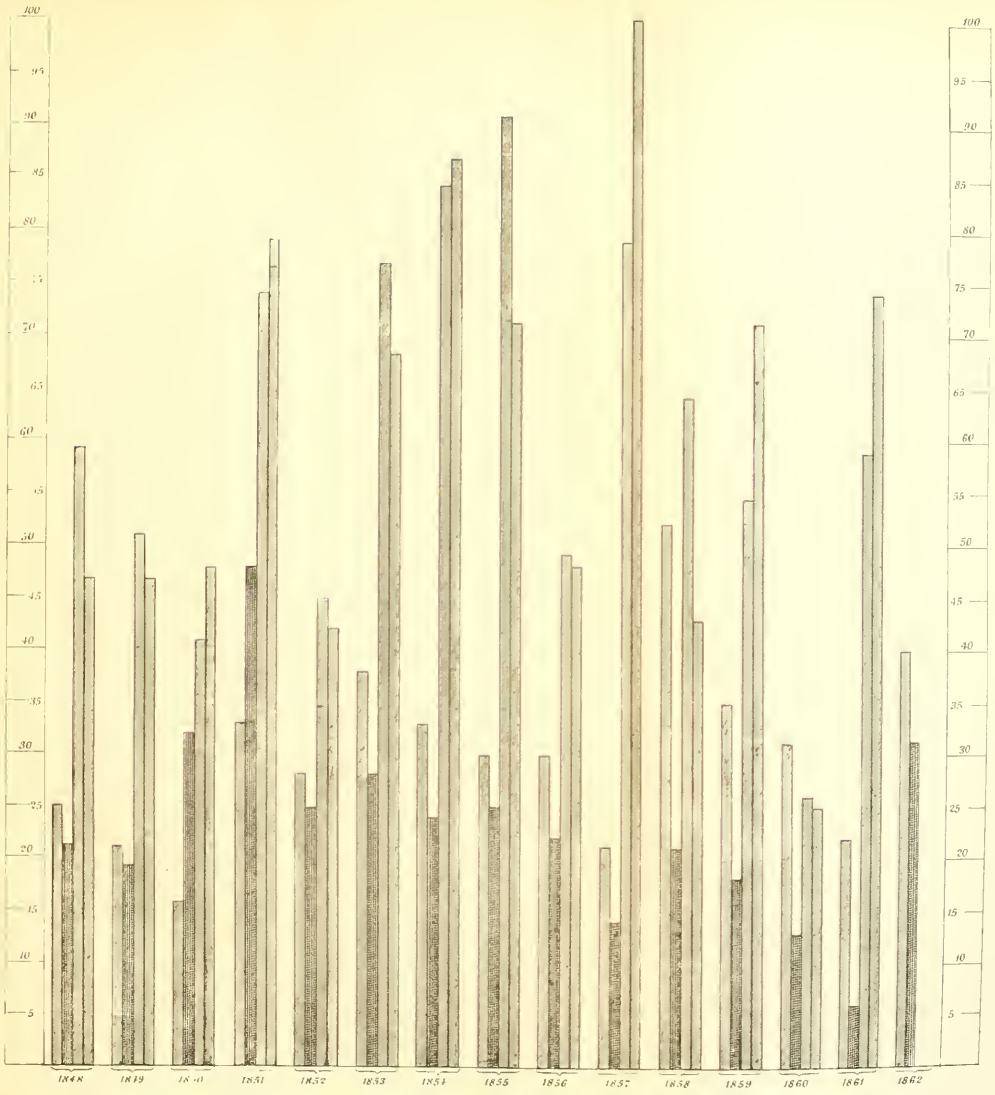

Diagram VIII shows the Quarterly admissions of Pythogenic Fever into the London Fever Hospital during fifteen years. (Compare with Diag III.)

Table XXXI.

Pythogenic Fever.—Months and Seasons.[m]

Years.	1848	1849	1850	1851	1852	1853	1854	1855	1856	1857	1858	1859	1860	1861	Total.	1862
January	9	9	6	13	10	17	13	16	12	8	32	13	14	8	180	17
February	9	7	5	8	12	7	13	9	10	5	7	10	6	8	116	6
March .	7	5	5	12	6	14	7	5	8	8	13	12	11	6	119	17
April .	4	3	7	8	7	5	6	8	8	4	5	5	7	3	80	4
May .	4	4	14	16	9	6	8	10	7	1	9	5	4	1	98	5
June .	13	12	11	24	9	17	10	7	7	9	7	8	2	2	138	25
July .	16	16	15	29	4	11	15	25	7	19	13	12	7	10	199	
August .	17	16	13	18	22	33	20	40	28	26	29	20	10	18	310	
Sept. .	26	19	13	27	19	33	49	26	14	34	22	20	9	31	342	
October	17	25	17	24	12	29	51	25	15	38	20	33	6	30	342	
Nov. .	19	16	17	30	12	26	20	22	28	33	15	27	6	31	302	
Dec. .	11	6	14	25	18	13	16	24	5	29	8	11	13	13	206	
Spring .	20	15	17	28	25	26	26	22	26	17	25	27	24	17	315	
Summer	33	32	40	69	22	34	33	42	21	29	29	25	13	13	435	
Autumn	60	60	43	69	53	95	120	91	57	98	71	73	25	79	994	
Winter .	39	31	37	68	40	56	49	62	45	70	55	51	33	52	688	
Total .	152	138	137	234	140	211	228	217	149	214	180	176	95	161	2432	

[m] See Note [1], page 65.

25 in summer; 51 in autumn; and 34 in winter.[n] Thirty-five years ago, Dr. Burne stated that there was no evidence of intestinal disease in the Continued Fevers of London, 'except in autumn.'[o] Most of the outbreaks of pythogenic fever in the provincial towns and villages of England, which have been recorded in the medical journals during the last twenty years, have occurred during autumn;[p] while the 'autumnal fever,' observed by Sir John Pringle and Rutty, in Britain and Ireland, during the last century, was apparently the same disease.[q] At Glasgow, in 1836 and 1837, Dr. Stewart observed that the cases of 'typhoid fever,' admitted into the Infirmary, were very numerous in the latter part of summer and in autumn, very few in winter and spring.[r] During September, October, and November, 1857, 18 cases were admitted into the Edinburgh Royal Infirmary; but in the three spring months of the same year, only 6 cases.

Similar observations have been made on the continent. At Geneva, M. Lombard, long ago, observed that the disease was always most prevalent in autumn.[s] Messrs. Rilliet and Barthez remark:—'Les nouveaux faits que nous avons recueillis con-'cordent avec les conclusions auxquelles sont arrivés MM. les 'docteurs Marc D'Espine et Lombard, savoir, que l'automne est 'de toutes les saisons, celle qui prédispose le plus à la fièvre 'typhoïde. Les trois épidémies qui ont spécialement atteint les 'enfants dans le canton de Genève ont toutes eu lieu en automne. 'Après l'automne vient l'hiver.'[t] Of 452 cases observed by Piedvache, during ten years, in the provinces of France, 316 occurred in autumn and winter, and only 54 in spring.[u] Of 116 circumscribed epidemics, which occurred in different parts of France, between 1841 and 1846, 20 commenced during the first quarter of the year, 21 during the second, 39 during the third, and 36 during the fourth.[v] In the 'Department du Doubs,' Druher says, that the disease is always most common in autumn and winter.[x] Of 183 cases at Strasbourg, reported by Forget, 60 occurred in autumn, 49 in summer, 38 in spring, and 36 in winter.[y] At Berlin, I am informed by Dr. Quincke, one of the physicians

[n] *Brit. & For. Med. Chir. Rev.* Oct. 1860.
[o] Burne, 1828, p. 129.
[p] See Edmondstone, 1818; *Bibliogr.* for 1846; *The Croydon Fever, Bib.* 1852; Beadle, 1853; Camps, 1855; Budd, 1856 (No. 1); Murchison, 1859 (No. 3).
[q] Pringle, 1752, p. 226; Rutty, 1770, pp. 196, 202, 320.
[r] Stewart, 1840, p. 291.
[s] Lombard, 1839 and 1843.
[t] Barthez and Rilliet, 1853, ii. 715.
[u] Piedvache, 1850, p. 20.
[v] Gaultier de Claubry, 1849, p. 8.
[x] Druher, 1858.
[y] Forget, 1841, p. 409.

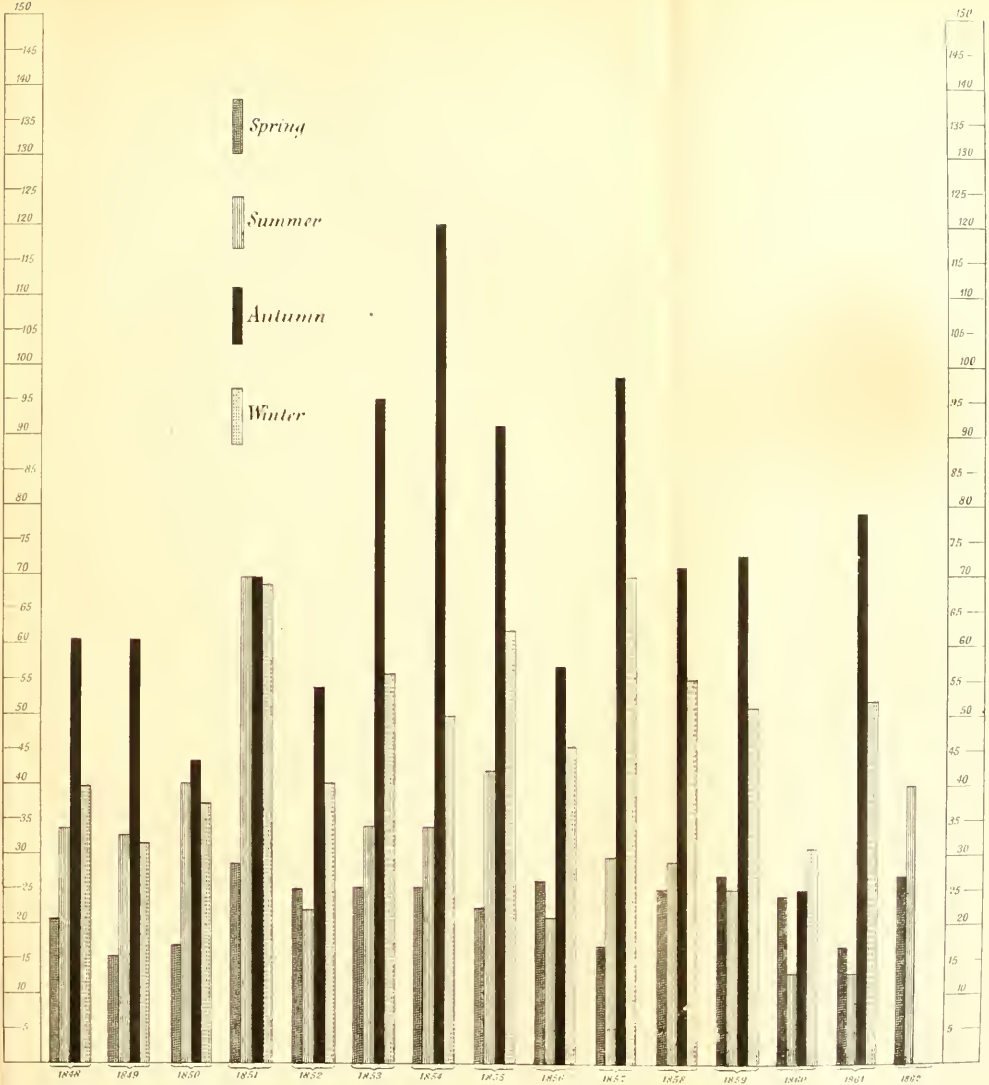

Diagram IX. shows the number of admissions of Pythogenic Fever into the London Fever Hospital, during each Season of fifteen years. Compare with Diag. IV.

to the Charité Hospital, that the disease is always most prevalent in autumn, and least prevalent in spring.

In America, Bartlett states that his impression is, that pythogenic fever is most prevalent in autumn. Of 645 cases admitted into the Lowell Hospital, during seven years, 250 were in autumn; only 104 in spring.[z] Wood says, that 'it is always most common 'in autumn and winter;'[a] while Austin Flint remarks, that in New England, it exhibits such a manifest predilection for the autumn, that it is there designated 'Autumnal or Fall Fever.'[b]

5. *Temperature and Moisture.*

Not only does pythogenic fever increase in frequency in autumn, but it has been found to be unusually prevalent after summers remarkable for their dryness and high temperature, and to be unusually scarce in summers and autumns which are cold and wet. The summer and autumn of 1846 were remarkable for their great heat, and the medical journals contain accounts of numerous outbreaks of enteric fever in various parts of the country-districts of England,[c] where the subsequent epidemic of typhus never appeared. Even in France, which was also not visited by typhus, 'typhoid fever' was unusually prevalent in the autumn of 1846, and was attributed by many to the excessive heat.[d] The Report of the London Fever Hospital for that year states, that 'in the 'unusually hot weather that prevailed in the summer and autumn 'months, diarrhœa occurred in almost every case of fever,' and that in the fatal cases, 'the intestines were extensively diseased.' It is not surprising, then, that an unusually large number of cases should have been observed at Edinburgh in the autumn and winter of 1846-7. As I have already endeavoured to show, this outbreak was independent of the great epidemic of typhus which immediately succeeded (see pages 50 and 76). Many instances will be alluded to hereafter of outbreaks of enteric fever occurring after very hot weather; but for the present it is sufficient to observe, that if a very hot season happen during an epidemic of typhus, both typhus and enteric fever may be unusually prevalent at one time, without necessitating the inference that both spring from a common origin. This was, possibly, the explanation of the slight increase of enteric fever observed towards the end of the typhus epidemic of 1826-8 (see page 44); at all events, the

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[z] Bartlett, 1856, p. 101.
[a] *Treat. on Pract. Med.* 4th ed. i. 389.
[b] Flint, 1852, p. 20.
[c] See *Bibliography* for 1846.
[d] De Claubry, 1849, pp. 18, 60.

summer and autumn of 1828 are said to have been remarkably hot. In 1837, Cless collected the records of all the outbreaks of enteric fever, which had occurred at Stuttgart, from 1783 to that date: all occurred in autumn or at the end of summer, and all had been preceded by unusually hot seasons. [e]

On the other hand, there have been few years in which the summer and autumn have been more cold and wet in England than in 1860, while the remarkable diminution in the prevalence of enteric fever over the whole country was a subject of general observation. On referring to Table XXXI., it will be seen that the admissions into the Fever Hospital, for that year, fell to one-half of the average of the other thirteen years, and that this diminution was due to the absence of the ordinary autumnal increase.

It may be doubted, if mere dryness of the atmosphere is conducive to an increase of enteric fever. The increased rainfall, however, sweeps away those impurities, to which the origin and spread of the disease are believed to be due.

6. *Intemperance, Fatigue, and Mental Emotions.*

There is no evidence that they predispose to pythogenic fever. In France, drunkards are said to be not more liable to the disease than temperate persons (see pages 69 and 307).

7. *Previous Diseases.*

It is doubtful if previous illness increases the liability to pythogenic fever. Most patients have been in good health at the time of seizure.

It is necessary, however, here to allude to certain relations supposed to subsist between pythogenic fever, on the one hand, and variola, malarious fevers, and phthisis, on the other.

Several recent French writers,[f] opposed to vaccination, have endeavoured to show that the practice has effected no reduction, but only a 'displacement of mortality,' and that, although smallpox has been arrested, it has been replaced by enteric fever, which, according to them, is nothing more than an internal variola, the eruption being developed in the intestines, instead of on the skin. It has been even proposed to the French Academy, to prevent pythogenic fever by vaccinating some portion of the mucous membrane. The subject was investigated with the utmost care by the French Academy, and the result was a complete refutation of M. Carnot's doctrine. 1. Pythogenic fever was not unknown, before

[e] Cless, 1837. For other illustrations of the increase of pythogenic fever in seasons of excessive heat, in France, see De Claubry, 1849, p. 18.

[f] Ançelon and Bayard, 1851; Gressot. 1855; Carnot, 1856.

the introduction of vaccination, as has been stated, but was as prevalent then as it is now. 2. It is only in exceptional cases, that the intestinal lesions of pythogenic fever bear any resemblance to the pustules of variola; and even then, the two morbid conditions are essentially different in their nature. 3. An attack of one disease confers no immunity from an attack of the other. Many instances have been observed, where persons have been attacked with pythogenic fever, immediately after having had small-pox, and where convalescents from pythogenic fever have been seized with small-pox. 4. The few vaccinated persons, who suffer on exposure to the variolous poison, are attacked with small-pox, although, according to M. Carnot's doctrine, when the variolous poison acts on the vaccinated body, the result should be pythogenic fever.[g]

Of even greater interest is the antagonism supposed to exist between pythogenic fever and the malarious fevers. The opinion has long been prevalent in America, that pythogenic fever has a tendency to take the place of intermittents and remittents, as these diseases, from the effects of cultivation and other causes, decrease or disappear.[h] This opinion has been, to some extent, corroborated by the investigations of M. Boudin,[i] who has endeavoured to demonstrate an antagonism between the diseases in question. According to this writer, localities where the constitution of the inhabitants is modified by malaria, are remarkable for the rarity of pythogenic fever, while localities remarkable for the prevalence of pythogenic fever, are likewise noted for the rarity and mildness of intermittents. Thirdly, he states, that the drying up of a marsh or its conversion into a lake, diminishes or arrests intermittents, but disposes the system to a new group of diseases, of which pulmonary phthisis and pythogenic fever are the most prominent; and fourthly, he maintains that, by residing in a marshy country, an individual acquires an immunity from pythogenic fever, the degree and duration of which are in direct proportion to the length and degree of the residence. M. Boudin mentions some remarkable instances of French regiments, which, after a lengthened exposure to malaria in Algeria, returned to France, where they remained exempt from pythogenic fever, although many cases were occurring in other regiments quartered in the same barracks. But it is doubtful, if any *antagonism*, such as M. Boudin has endeavoured to establish, really exists between intermittents and pytho-

[g] On this subject, see Mr. able Simon's Report on Vaccination, 1857, p. 56; also, Barthez and Rilliet, 1853, iii. 63.

[h] Bartlett, 1856, p. 100. [i] Boudin, 1846.

genic fever. The latter is not unknown in countries remarkable for the prevalence of intermittents: it is not uncommon in India, Burmah, and other malarious countries, where it has probably been often *mistaken for* remittent fever. The facts mentioned by American writers, and by M. Boudin, suggest a *similarity*, rather than an *antagonism* of pythogenic and malarious fevers, the poisons in both instances being generated under similar circumstances. In connection with this subject, a remarkable communication made to the French Academy of Sciences in 1845, by M. Ançelon, may be mentioned.[k] Many years before, pythogenic fever had been constantly endemic in the Commune of Guermange, in the duchy of Lorraine, making its appearance every year during the hot season; but, for twenty-five years, it had entirely disappeared from the northern part of the commune, its disappearance having been simultaneous with the suppression of a stagnant pond in that locality. At the southern part of the commune, however, there had been epidemics of intermittent fever every third year (viz., 1829, 1832-5-8, 1841,) alternating with epidemics of pythogenic fever (1830-3-6-9, 1842), and of furuncular diseases (1831-4-7, 1840-3). At this part of the commune there was a large lake called the 'Indre basse,' which every third year, was emptied and cultivated, and afterwards the water was allowed to collect again for two years. The intermittent fevers appeared during the first year that the pond was full of water. The epidemics of pythogenic fever coincided with the second year. In the autumn of this year the pond began to dry up, and M. Ançelon attributed the fever to the action of heat and moisture upon an immense quantity of *animal* and vegetable *débris*, which during the two years had been collecting upon the banks of the lake. The houses in the commune were also very badly drained. This is far from being a solitary instance. M. Killiches has recorded an outbreak of pythogenic fever, which occurred at a small town of Bohemia, on the drying up of a lake.[l] Other instances will be found in the Reports on Epidemics to the French Academy.[m]

According to M. Forget, persons labouring under phthisis are rarely attacked with pythogenic fever. The former he regards as a preservative against the latter.[n] Whether this be so or not, an attack of pythogenic fever is often followed by tubercular deposit in the lungs.

Lastly, it is maintained by Stöber, Löschner, and Friedleben,[o]

[k] Ançelon, 1845.
[l] Killiches, 1837. [m] De Claubry, 1849, p. 54. [n] Forget, 1841 p. 331.
[o] *Brit. & For. Med. Chir. Rev.* July, 1858, p. 162.

that pythogenic and scarlet fevers, have an inverse ratio, as regards epidemic prevalence, and that when one prevails, only solitary cases of the other are to be met with. My experience is opposed to their observations. I have often noticed the two diseases unusually prevalent at the same time. It was so at Windsor, in 1858; and during 1861, 123 out of 161 cases of pythogenic fever, and 123 out of 173 cases of scarlet fever, were admitted into the London Fever Hospital in the last five months of the year. The returns of the Registrar-General also show, that the mortality from scarlet fever is always greatest at the end of autumn, the time at which pythogenic fever is most prevalent.

8. *Idiosyncrasy.*

There are many facts which seem to show that certain peculiarities of constitution favour or avert an attack.

9. *Over-crowding and Deficient Ventilation.*

The prevalence of pythogenic fever is independent of overcrowding and deficient ventilation. The disease prevails without distinction, not only in the most dense, but also in the least populous, districts of large towns, and is of common occurrence in country-districts, and even in isolated houses. As typhus and relapsing fever prevail only in crowded localities, and pythogenic fever in all, it follows, that in the central and most crowded districts of the metropolis, the number of cases of the former far exceeds that of the latter; but, on passing to the suburban districts, the proportion of pythogenic cases gradually increases, while in the country, they constitute almost the sole fever met with. This appears, to some extent, from the residences of the patients brought to the London Fever Hospital, given in Table IX., p. 72, of which the following is an abstract:—

Table XXXII.

DISTRICTS.	Population in each statute acre, in 1851.	Typhus and Relapsing.	Pythogenic.
Central—			
Holborn - - -	238	608	133
City of London - -	200	508	91
St. George's-in-the-East -	200	372	100
Suburban—			
Paddington - - -	35	1	12
Hackney - - -	15	22	53
Beyond London Districts-	?	8	36

Paddington and Belgravia are two of the least populous London districts, and, at the same time, are inhabited by the better classes of the community. Now, of 6 cases of fever from Belgravia, 4 were pythogenic and 1 typhus; and of 14 cases from Paddington, 12 were pythogenic and 1 typhus. That pythogenic is the prevailing fever in each of these districts, is also shown by the cases admitted into their local hospitals. By the published reports of St. George's Hospital,[p] situated in Belgravia, it appears that of 44 fatal cases of fever dissected during three years, there was ulceration of Peyer's patches in 29, and in 5 only were the intestines healthy. With regard to Paddington, the following table shows the forms of fever admitted into St. Mary's Hospital during five years from that parish. From this table, all the cases of fever admitted from other districts, except Paddington, have been excluded:—

TABLE XXXIII.

	1853.	1854.	1855.	1856.	1857.	Total.
Typhus	...	1	1?	2	1	5
Pythogenic.....	15	20	12	14	14	75
Febricula	1	6	5	3	5	20
Doubtful cases	11	1	1	1	3	17
Total	27	28	19	20	23	117

It appears, then, that during five years, 75 cases of pythogenic fever were admitted from Paddington into St. Mary's Hospital, and only 5 of typhus; and that, in the year 1856, only 2 cases of typhus were admitted, while in the same year, there were admitted into the London Fever Hospital, 1,062 cases.

The fact that pythogenic fever is independent of over-crowding, has been a matter of general observation. For many years, most of the cases admitted into the Glasgow[q] and Edinburgh[r] Infirmaries were brought from the localities in the neighbouring country, and not from the crowded parts of the town, to which the cases of typhus were restricted. Bartlett observes, that there is no satisfactory evidence that over-crowding predisposes to this fever in America;[s] and with respect to Paris, Louis remarks:—
'Le séjour dans les lieux bas et habités par un trop grand nombre
'de personnes, pendant la nuit, ne peut pas non plus figurer parmi
'les causes dont il s' agit.'[t]

But though pythogenic fever is far from being limited to crowded

[p] Vide *Brit. & For. Med. Chir. Rev.* 1855-6. [q] STEWART, 1858.
[r] REID, 1842. [s] BARTLETT, 1856, p. 110. [t] LOUIS, 1841, ii. p. 356.

localities, it is probable that deficient ventilation may occasionally favour the action of the poison, by preventing its diffusion and dilution.

10. *Recent Residence in an Infected Locality.*

Petit and Serres,[u] and afterwards Andral,[x] Louis,[y] and Chomel,[z] strongly insisted on recent residence as a predisposing cause of pythogenic fever. Andral noticed that medical students were most liable to be attacked within a few weeks of their arrival in Paris. Of 129 cases, which Louis gives in his work, 73 had not resided in Paris more than ten months, and 102 not more than twenty months. Again, of 92 cases of 'typhoid fever' under Chomel, in the Hotel Dieu, one-half had resided in Paris only one year, or less. The length of residence in London of all the cases of pythogenic fever admitted into the London Fever Hospital, during fourteen years, where the circumstance was noted, was as follows :—

TABLE XXXIV.

Less than 3 months	122	or	6·17 per cent.
,, 6 months	191	,,	9·65 ,,
,, 1 year	318	,,	16·07 ,,
,, 2 years	432	,,	21·84 ,,
,, 10 years	771	,,	38·98 ,,
More than 10 years, but not for life	149	,,	7·53 ,,
For entire life	1,058	,,	53·49 ,,
Total	1,978	,,	100· ,,

Upwards of six per cent. of the patients had not resided in London three months before the date of their admission into Hospital. This circumstance does not admit of the explanation offered in the case of relapsing fever. It has been already pointed out that the newly-arrived patients did not come from Ireland. Almost all of them came from the provinces of England, and were in good health and comfortable circumstances at the date of their arrival in London. Many of them were servants in private families. Moreover, the above figures are far from indicating, to its full extent, the influence of change of residence in predisposing to pythogenic fever. A large proportion of the patients were first attacked within a few days or weeks after changing their place of residence in London. I have also known several instances where successive visitors at the same house, at intervals of months, or

[u] PETIT and SERRES, 1813, p. 127. [x] ANDRAL, 1823, ed. 1833, i. 484.
[y] LOUIS, 1841, ii. 357. [z] CHOMEL, 1834.

even years, have been seized, shortly after their arrival, with pythogenic fever, or diarrhœa, from which the ordinary residents were exempt. These considerations point to the dependence of pythogenic fever on some local cause, to which the system becomes habituated by constant exposure.

11. *Occupation.*

The occupation of 1457 cases of pythogenic fever admitted into the London Fever Hospital is stated in Table VII. (page 68). It is not probable that any of the occupations specified, in themselves predispose to the disease. It will be noticed, however, that a large proportion, nearly one-third, were female servants, most of whom were in comfortable situations, and many of whom were attacked shortly after changing their residence. It may also be mentioned that several of the patients, entered as 'labourers,' had been engaged in the public sewers before their seizure. Of 64 vagrants, 44 had typhus, 12 relapsing fever, and 8 febricula, but not one pythogenic fever. Of 247 hawkers and street musicians, 136 had typhus, 54 relapsing fever, and only 24 pythogenic fever. On the other hand, of 45 policemen, 30 had pythogenic fever, 10 typhus, and 5 febricula, but none relapsing fever.

12. *Station in Life.*

Destitution does not predispose to pythogenic fever. Indeed I am inclined to think that persons in good circumstances are more liable to it than the poor. While epidemics of typhus and relapsing fever invariably commence among the poorest of the population, and are, for the most part, confined to this class, it has been a common observation in almost every epidemic of pythogenic fever that the rich have not remained exempt, and in many instances the epidemic has commenced among the upper classes. At Nottingham, in 1846, Dr. Sibson remarked that 'very many were in good circumstances of those who were attacked:'[a] at Croydon, in 1852, we are told that the victims were 'not among 'the poor, but among the gentry and principal tradesmen of the 'town:'[b] at Windsor, in 1858, the fever was confined, for the most part, to the upper and middle classes; the poorest and worst part of the town, to a great extent, escaped.[c] In fact, pythogenic fever is far from being an uncommon disease among the upper classes in England, and recent events have shown that the most exalted positions offer no protection from it. Similar observations have been made in America by Bartlett,[d] and in France by Andral,[e]

[a] Sibson, 1846. [b] See *Croydon*, 1852. [c] Murchison, 1859 (No. 3).
[d] Bartlett, 1856, p. 110. [e] Andral, 1823, ed. 1833, i. 484.

Louis,[f] Piedvache,[g] and other observers. Indeed, the evidence on the point is overwhelming. The contrast which pythogenic fever exhibits to typhus and relapsing fever, in this respect, is borne out by the experience of the London Fever Hospital. The patients admitted into this institution may be divided into four classes, viz.:—1. The servants of subscribers, policemen, and persons able to pay for admission. Exceptional cases are included in this class where destitute persons are paid for by the charitable. 2, Free patients, not receiving parochial relief. This is a mixed class: some have been destitute, while others have been in easy circumstances up to their illness. 3. Patients paid for by the parishes, but not inmates of a workhouse. 4. Inmates of workhouses. These classes represent four different grades in worldly comfort, and the following Table shows the proportion of the different fevers in each class during ten years (1848-57).

TABLE XXXV.

	Class I.			Class II.			Class III.			Class IV.		
	Number.	Per centage of each fever, on total of Class I.	Per centage of cases in Class I., on total of each fever.	Number.	Per centage of each fever, on total of Class II.	Per centage of cases in Class II., on total of each fever.	Number.	Per centage of each fever, on total of Class III.	Per centage of cases in Class III., on total of each fever.	Number.	Per centage of each fever, on total of Class IV.	Per centage of cases in Class IV., on total of each fever.
Typhus....	94	22·06	2·68	130	29·75	3·7[5]	2,544	53·26	72·56	738	74·62	21·05
Relapsing..	2	·47	·45	9	2·06	2·04	383	8·02	86·85	47	4·75	10·65
Pythogenic	281	65·96	15·44	245	56·06	13·46	1,209	25·31	66·	85	8·59	4·67
Febricula .	49	11·5	5·69	53	12·13	6·15	640	13·4	74·33	119	12·03	13·82
Total.....	426	99·99	6·43	437	100·	6·59	4,776	99·99	72·06	989	99·99	14·92

In Class I. the proportion of pythogenic cases is six times that of typhus and more than thirty-four times that of relapsing fever. In Class II. the proportion of the pythogenic cases is still predominant, although to a less extent, being four times that of typhus, and six times that of relapsing fever. In Classes III. and IV. the proportion is reversed, typhus and relapsing fever being in excess of pythogenic. The contrast presented by pythogenic fever to relapsing fever and typhus appears from the following comparison:—

TABLE XXXVI.

	Per cent. of Typhus and Relapsing.	Per cent. of Pythogenic.
Of the paying patients . . .	22·5	65·96
Of the 'free' patients . . .	31·8	56·06
Of those sent from homes by parishes	61·28	25·31
Of the inmates of workhouses .	79·37	8·59

[f] LOUIS, 1841, ii. 356. [g] PIEDVACHE, 1850, p. 21.

B. Exciting Causes.

1. *Contagion.*[h]

While it has been almost universally admitted that typhus and relapsing fever are eminently contagious, many of the best authorities have entertained grave misgivings as to the communicability of pythogenic fever. Andral, in 1833, declared that he had never seen it exhibit the slightest contagious character, either in hospital or private practice;[i] and, in the following year, Chomel stated that not more than one in a hundred medical men in France believed it to be contagious.[k] In 1840, Dr. Stewart wrote as follows:—'In no case, though questioned with the greatest care, 'either in Scotland, or in the hospitals of Paris, have I ever found 'the disease referred to contagion.'[l]

Certain French observers, however, have recorded many facts to prove that 'typhoid fever' is communicable. Leuret, in 1828,[m] endeavoured to show that its introduction into Nancy was due to contagion; and in the subsequent year, Bretonneau communicated to the 'Académie de Médecine,' a number of observations, with the object of proving that 'dothiénentérie,' as it prevailed in the country, was eminently contagious.[n] These essays were followed in 1834 by the memoir of M. Gendron, of Château du Loir,[o] who maintained that every case was due to contagion, and that 'typhoid 'fever' ought to be ranked amongst the most contagious maladies. Many additional facts, tending to prove its contagious nature in country districts were subsequently recorded by various writers.[p] These observations excited much discussion, physicians in Paris still maintaining, that in that city the disease rarely spread by contagion, whatever might be the case in the provinces. Even Louis, in 1841, while fully admitting the facts recorded by Bretonneau, Gendron, and others, stated, that in his extensive experience he had only met with three instances, in which the disease could be said to have originated from contagion.

In 1849, appeared the Prize Essay of M. Piedvache, of Dinan: '*Recherches sur la contagion de la fièvre typhoïde.*'[q] In this essay many facts noted by the author, and recorded by previous observers, were collected; the evidence on both sides of the question was honestly weighed, and the conclusion was arrived at that the disease was contagious, but only under certain conditions, while, at the same time, it was admitted, that many facts

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[h] See note, page 79. [i] Andral, 1833, i. 485. [k] Chomel, 1834.
[l] Stewart, 1840, p. 298. [m] Leuret, 1829. [n] Bretonneau, 1829.
[o] Gendron, 1834. [p] See *Bibliography*, 1834 to 1847. [q] Piedvache, 1850, p. 72.

' prouvent évidemment que ce phénomène (contagion) n' a pas ' toujours lieu.' The same view is adopted by Trousseau.[q]

In America, and in Britain, opinions are also divided on the subject; but most observers now believe, that although the disease is communicable in a limited degree, it is impossible, in many cases, to discover any source of contagion. In England, as in France, there are writers who hold extreme views, some believing that there is no conclusive evidence to show whether the disease be in any way contagious, while others maintain, as Dr. W. Budd has so zealously done, that the contagious nature of enteric fever is the 'master truth' in its history.[r]

The question is of such importance, that a consideration of the chief arguments in favour of the contagious nature of the disease may be advantageous.

a. When one individual is attacked, many other cases often follow in succession in the same house or district. Facts of this nature are common in both town and country-districts; but undue stress has been laid on them by the advocates of contagion. A moment's reflection shows that such cases are as readily explicable, on the supposition that the disease has a local origin, as upon that of contagion. Although, in some instances, the cases follow one another, so as to favour the idea that the disease has been communicated by one patient to the other, the circumstances in others are rather opposed to such a view. Occasionally, many persons residing in one house, even as many as twenty or forty, are seized all at once, so as to suggest the suspicion of poisoning, and yet no source of contagion can be traced. On the other hand, the interval between the different cases, sometimes appears too long, to admit of explanation, on the theory of contagion. I have met with several instances, where single cases of pythogenic fever have originated in the same house year after year, without any traceable importation of the poison on any occasion. For instance, six cases were admitted from a single house into the London Fever Hospital; one in June, 1849; one in October, 1851; one in February, 1854; one in November, 1855; one in November, 1856; and a sixth in July, 1857. Moreover, the order of succession of the cases has often no relation to the degree of exposure to the supposed source of contagion. Piedvache mentions a remarkable instance of pythogenic fever in a boys' school, at Dinan. The boy first attacked was nursed by his fellow-pupils, more than twenty of whom passed the night with him during his illness, and used no precaution

[q] Trousseau, 1861. [r] W. Budd, 1856, 1859, 1861.

against the contagion. Not one of the boys thus exposed took the fever; but the second case occurred nineteen days after the death of the first, in a boy who had no communication with the first patient, who had never entered his room, and who slept in a remote part of the building.[s]

b. Pythogenic Fever is said to be communicated to the nurses and other attendants on the sick. Many instances might be cited where nurses, who have gone to attend on patients suffering from pythogenic fever at their own homes, have been attacked shortly after their arrival; but, on the supposition that the disease may have a local origin, the nurse is exposed to the poison equally with the residents, and, in fact, the recent date of her exposure renders her more liable. I have never known or heard of a case, where the fever has been communicated to the medical attendant not residing in the infected house; and Piedvache makes a similar statement.[t] It is, therefore, necessary to have recourse to evidence derived from what occurs when patients are treated in different localities from those in which they contracted the disease.

Hospital experience lends little support to the doctrine of contagion. One of the chief arguments for the contagious character of typhus was derived from the liability of hospital attendants to suffer; but it is universally admitted to be a very rare occurrence for the nurses or medical attendants of hospitals to contract pythogenic fever from the sick under their care. Andral denied that it was ever communicated to the medical attendants in a hospital, or to patients occupying adjoining beds.[u] During six years, not a single case of contagion occurred in the *clinique* of M. Bretonneau, at Tours.[x] Louis, in his extensive experience at the hospitals of La Pitié and the Hotel Dieu, met with only three instances where the disease originated in these institutions.[y] During nineteen years, Chomel only knew four cases contracted in the wards of the Hotel Dieu;[z] and Piedvache, as the result of his extensive research, declared that, in France, such cases were quite exceptional.[a] Dr. Peacock remarked that he had never known pythogenic fever communicated to the nurses and attendants at St. Thomas's Hospital;[b] and, from repeated inquiries, I am in a position to state that, in every general hospital of the metropolis, such an occurrence is extremely rare. Since my connection with the London Fever Hospital, 1,048 cases of pythogenic fever have been treated

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[s] Piedvache, 1850. [t] Ibid. p. 93.
[u] Andral, 1833, i. 485. [x] De Claubry, 1845, p. 844. [y] Louis, 1841, ii. 374.
[z] Chomel, 1834. [a] Piedvache, 1850, p. 84. [b] Peacock, 1856.

in the hospital, but in only two instances has the disease appeared to originate there; yet, in the same period, 36 of the attendants and patients have contracted typhus, the number of patients admitted with typhus being 2,581. During the last 14½ years (1848—62), 2,506 cases of pythogenic fever have been admitted into the hospital, and 8 cases are reported as having originated there. They are as follows:—Nurses, 2 cases; servants, 2; patients admitted with typhus, 2; patients admitted with scarlatina, 2: total, 8 cases.

This number is certainly small, when compared with the number of typhus cases originating in the institution during the same period (see page 81), and is, perhaps, not greater than might be expected during fourteen years in a varying population of upwards of a hundred persons, outside the hospital. At the same time, it would be difficult to account for their origin otherwise than on the supposition of contagion. This also would appear to be the only explanation applicable to the following instances of pythogenic fever, contracted by the attendants on the sick:—

1. In the year 1858, one of the nurses at the King's College Hospital, between twenty-five and thirty years of age, contracted well-marked pythogenic fever and died. Immediately before her seizure, she had been engaged in nursing a patient ill of the disease. None of the other nurses or of the patients in the hospital caught the fever, which could not therefore be supposed to have had a local origin.

2. A similar case is recorded by Gendron. On November 5th, 1826, a female, aged twenty, was brought to the hospital of Château-du-Loir. She was then in the third week of an attack of 'dothinenteritis,' of which she died on December 1st. Immediately after her death, her nurse, a female aged forty-five, was attacked with the fever, no other cases of which occurred in the hospital.[c]

3. Some years ago, two young men met in London. A came from the Isle of Wight, where there was no fever; B came from a village in Cambridge, where pythogenic fever was prevalent. B was ill at the time of meeting. Both proceeded to Edinburgh, where B had a well-marked attack of pythogenic fever. A lived in the same house and nursed B, and he also took the fever, although all the other residents in the house escaped.[d]

4. At Windsor, in 1858, Emily C—— was brought home ill of pythogenic fever to her father's house. She was nursed by her

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[c] PIEDVACHE, 1850, p. 50; see also page 52.
[d] Communicated by Dr. BUCHANAN.

sister Amelia, aged twelve, who slept in the same room on a mattress beside her sister's bed. At the end of a fortnight, Amelia was seized with the fever, which ran a severe course, and presented all the characteristic symptoms, including the lenticular spots and diarrhœa. Pythogenic fever was certainly prevalent in the neighbourhood; but though several of the residents in the same house were of the age most liable to it, Amelia C——, who alone attended on her sister, was the only one who took the fever.[e]

c. Persons labouring under pythogenic fever, sometimes transport it into localities, where it was before unknown, but where it then spreads from them as from a centre. Although many of the cases appealed to in support of this argument, have probably been examples of typhus, or of some other fever,[f] it cannot be doubted that there are unequivocal instances of pythogenic fever, which appear to be propagated in the manner described. It may be true, that such occurrences are exceptional, and that the number of cases where the disease is introduced into a new locality without spreading, exceeds that in which it is propagated. It is also probable, that the fever is occasionally believed to be introduced into a house by a newly-arrived servant, when it really has a local origin, from which the servant naturally suffered first. In several instances of this sort, I have ascertained that the servant was perfectly well at the time of arrival. Such arguments, however, do not invalidate the conclusion to be drawn from cases where the disease has spread in a circumscribed locality, immediately after the arrival of an infected person. The following illustrations are to the point.[g]

1. In 1826, an outbreak of pythogenic fever occurred in the Military School of La Flèche, in France. It commenced in July, and did not cease until 109 boys were attacked. The school was broken up, and the boys who were not ill were sent to their own homes in distant parts of France ; 29 were taken ill after reaching their homes, and 8 communicated the disease to their families.[h]

2. In 1858, a servant ill of pythogenic fever was removed from Windsor to her home at Cippenham, four miles distant. Three weeks afterwards, her father and sister took the disease, although no other cases had occurred at Cippenham. Another girl ill of the fever, was removed to Bray, some miles distant. Shortly

[e] Murchison, 1859 (3), p. 311.

[f] For example, at Windsor in 1858, most of the cases popularly reported as proving the contagious character of pythogenic fever, proved to be cases of scarlet fever, which was very prevalent at the same time.

[g] Others are reported by the writers already referred to (page 428) and also by Reeves (1859 ; Simon (1861); and Trousseau (1861).

[h] Bretonneau, 1829, p. 70.

after, her two sisters took the fever, although it was stated that no other cases had occurred at Bray previously.[i]

3. Dr. W. Budd has recorded an outbreak of pythogenic fever which occurred at North Tawton, Devon, in autumn, 1839. During the prevalence of the fever, it so happened that three persons left the place after they had become infected, and all three communicated the disease to one or more of the persons by whom they were surrounded in the new neighbourhoods to which they had removed, although, in each of the three new localities, there had been no cases of fever previous to their arrival.[k]

4. On the 16th of December, 1858, a lad about twelve years old was brought from a boarding-school, at Cardiff, in the first stage of pythogenic fever, to his father's home, which was a farm-house, situated on the crest of a hill, five miles to the west of Cardiff. Fever had not occurred at this farm within the memory of man. Before the arrival of the infected lad, the family were in the enjoyment of good health, and the neighbouring village and farms were entirely free from fever; yet, shortly after his arrival, three sisters, two brothers, two servants, and a hired nurse, all contracted the fever.[l]

5. Pythogenic fever broke out in a family living in an isolated country house on the top of a hill, in France. Three nurses were called in to tend the sick. All three took the fever, and all three communicated it to their own families, residing in a village at a long distance from the source of infection.[m]

6. A boy, aged twelve, was carried home ill with pythogenic fever. His home was an isolated house in the country, in France, nine miles distant from where the fever was prevailing. His family had before been in good health; but immediately after, his father, mother, and three brothers and sisters, were attacked by the fever.[n]

In the face of such facts, it is impossible to deny that pythogenic fever is communicable by means of some poison emanating from the sick. Before proceeding to discuss whether this poison be ever generated spontaneously, it may be expedient to allude to some of the laws by which its action appears to be regulated.

1. *Mode of Communication.* Although pythogenic fever be communicable, it is not so certain that it is contagious in the strict sense of the term. From what we know, it does not appear that mere contact with the sick is sufficient or necessary to produce it.

[i] Murchison, 1859 (3), p. 311. [k] Budd, 1859, p. 29. [l] Ibid, p. 30.
[m] Piedvache, 1850, p. 60. [n] Gendron, 1834.

It is not improbable, that, as in dysentery and cholera, the alvine dejections constitute the chief, if not the sole, medium of communication. This view, which has been strongly advocated by Dr. W. Budd,[o] and which has been taught at Munich for many years by Professor von Gietl,[p] would, if correct, account for some of the differences of opinion which have existed on the subject of contagion, as the disease would not appear to be communicable, when care was taken at once to remove and destroy the alvine evacuations, in which the specific poison is believed to reside.

This subject will be discussed at greater length under the head of Spontaneous Origin.

2. *The distance to which the poison can be transmitted.* Piedvache and other writers have maintained that the poison of pythogenic fever, like that of typhus, ceases to take effect at a very short distance from the sick, and that it is always inert when the atmosphere around the sick is constantly changed. Although deficient ventilation must prevent the diffusion of the poison, and render it more virulent, whether it proceed from infected persons or not, it is obvious, that if the poison be contained in the stools, it may take effect at a distance from the persons from whom it is derived.

3. *Fomites.* Bretonneau, Gendron, and other observers, have imagined that the poison of pythogenic fever can adhere to the clothes and bedding of the sick, and that the disease may thus be propagated. Gendron, who was an exclusive contagionist, cited several instances, where he believed that the disease was transmitted by bedding after an interval of many years;[q] but in these cases, the cause was probably localized in the house, and not in the bedding. I have never met with an instance which gave any support to the opinion that pythogenic fever can be propagated by fomites, and there are few contagionists who believe that this is possible. At the same time, it is right to allude to the following fact, communicated to me on excellent authority:—

In 1859, the wife of a butcher, residing in the small village of Warbstowe, situate between Launceston and Camelford, on the Cornish moors, travelled to Cardiff, in Wales, to see her sister who was ill, and soon after died of 'typhoid fever.' She brought back her sister's bedding. A fortnight after her return to Warbstowe, another sister was employed in hanging out these clothes, and soon after was taken ill with 'typhoid fever,' which spread from her as from a centre. The woman who had been to Cardiff never took

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[o] W. Budd, 1856, 1859, 1861. [p] Von Gietl, 1860, p. 2.
[q] Gendron, 1834; Piedvache, 1850, p. 119.

the fever herself; there had been no cases in Warbstowe previous to her return; neither were there any cases in the neighbouring villages, either before or after.

4. *Latent Period.* In many cases, there appears to be a period of incubation between exposure to the poison, and the commencement of the malady. In consequence of the insidious manner in which the disease often commences, it is more difficult than in the case of typhus, to determine the length of this period; but, as a rule, it appears to be between one and two weeks. Dr. W. Budd states, that a large number of facts have led him to the conclusion that it ranges from ten to fourteen days.[r] When the school of La Flèche was broken up on account of an outbreak of pythogenic fever (see page 432), of the 29 boys who fell ill with the fever at their own homes, all were seized some time during the second week after their arrival.

Cases have been reported, where the latent period has been thought to extend over many weeks or months;[s] but in all such cases, it may be doubted if the disease has not had a spontaneous origin.

On the other hand, if the poison be sufficiently strong, there is reason to believe that the seizure may be almost instantaneous. Under such circumstances, the disease is usually ushered in with vomiting and purging, and may attack at once many persons residing in the same house. It is not surprising, then, that many outbreaks of pythogenic fever have at first excited suspicions of poisoning. Many instances will be referred to presently, where the symptoms of pythogenic fever were at first ascribed to criminal or accidental poisoning, whilst the events which followed the late outbreak of pythogenic fever in the royal family of Portugal, must be fresh in the memory of all.[t]

5. *Stage at which the disease is most communicable.* There are no data for forming an accurate opinion on this point. According to Gendron and other writers, the disease is most contagious at its advanced stage; but this conclusion is merely founded on the circumstance that the first patient in a house has occasionally been ill for two or three weeks before the others are attacked. If the poison reside in the stools, it would be important to ascertain at what stage they are most virulent.

There is no proof that pythogenic fever can be communicated by the dead body. Putegnat was inclined to attribute his own

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[r] Budd, 1856, p. 618. [s] See Bree, 1846.
[t] See *Brit. Med. Journ.* Jan. 4th, 1862.

attack to the autopsy of a fatal case. It is true that he was seized a few days after the autopsy; but he had attended both the patient and her mother during their illness.[u] Féron cites the instance of a woman who went a distance of two miles to lay out the body of a little girl who had died of the fever, and who was herself seized immediately after; but the circumstance is equally explicable on the supposition of some local cause in the house where the girl had died.[x]

6. *Immunity from second attacks.* It is generally admitted, that one attack of pythogenic fever confers an immunity from subsequent attacks.[y] This opinion is founded on observations of a two-fold nature. First, on questioning patients suffering from the disease, it is rarely ascertained that they have had a previous attack. Former attacks of 'fever' have usually been of a different nature. Secondly, many remarkable instances have been recorded, particularly by Gendron and Piedvache, where a second outbreak has occurred in the same house or locality after an interval of many years, and where the fever has attacked almost every person who had not the disease previously, but spared all who were attacked in the first visitation.

At the same time, there are well-authenticated cases of persons contracting pythogenic fever a second time. Piedvache mentions the case of a girl who had an attack in January, 1841, at the age of ten, and a second attack in July, 1849[z]. Three unequivocal examples of a second attack are reported by Michel;[a] three by Bartlett, after an interval of only one year; and four by Dr. W. Budd. Such cases, however, are exceptional (see page 94.)

2. *Spontaneous Generation.*

Admitting that pythogenic fever is, under certain circumstances, communicable, I believe that it is equally true, that many cases have a spontaneous origin. Of the patients admitted into the London Fever Hospital, I have rarely been able to trace the disease to contagion. Of 1,576 cases, it was ascertained that 204, or 13·72 per cent., attributed the disease to contagion, but only because other cases had occurred in the same house (see page 429). Although in large towns, it may be difficult to exclude the possibility of contagion, on turning to the history of circumscribed epi-

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[u] Putegnat, 1838, p. 856.
[x] Féron, 1840, p. 105.
[y] Bretonneau, 1829, p. 58; Gendron, 1834; Chomel, 1834, p. 333; Louis, 1841, ii. 370; Piedvache, 1850, p. 103; Jenner, 1849 (1). 38; W. Budd, 1859 p. 56; Bartlett, 1856, p. 106.
[z] Piedvache, 1850, p. 103.
[a] Michel, 1859, p. 297.

demics in country-districts, it is found to be often impossible to attribute the first appearance of the disease to contagion. It is not uncommon for the inmates of an isolated country house to be seized with pythogenic fever, although no case has occurred within many miles, and there is no evidence of importation of the poison. In fact, if we except Bretonneau, Gendron, and our countryman, Dr. W. Budd, it has been almost universally believed, that a large proportion of the cases of pythogenic fever are independent of contagion. Even Gendron admitted, that, after the most rigid investigation, he was quite unable to account for the first cases in certain localities, and he added, that he had met with several isolated cases, of which the cause was unknown to him.[b] Piedvache also states, that, in France, it is often impossible to trace the first cases of a circumscribed epidemic to contagion, and records many instances, where it appeared certain that the persons attacked had not directly or indirectly been exposed to contagion. His conclusions on this point are as follows: 'Je dirai même qu'il est très 'probable, et je crois même certain, que des fièvres typhoïdes, dans 'quelques circonstances, se déclarent à la fois en nombre assez 'considérable pour constituer une épidémie indépendamment de la 'contagion.'[c] Dr. Wood, of America, remarks: 'But against the 'opinion of its ordinary contagiousness, is the fact, that it is con-'stantly springing up in isolated cases, without any possible com-'munication.'[d] 'La transmissibilité,' says Jacquot, 'est la règle 'pour le typhus, l'exception pour la dothiénentérie.'[e] Lastly, Trousseau, although a decided contagionist, admits, that, in many instances, its origin is spontaneous.[f]

Until a few years ago, it was not attempted to account for the spontaneous origin of enteric fever. Chomel remarked: 'Les causes 'de la fièvre typhoïde sont enveloppées de la plus grand obscurité.'[g] Dr. Stewart observed: 'With regard to the producing cause of 'typhoid fever, all is vague and uncertain.'[h] Piedvache spoke of its etiology as 'enveloped in obscurity;' and in March, 1858, Dr. Tweedie stated, in his lectures delivered before the Royal College of Physicians, that its causes were 'obscure and unknown.'

Emanations from sewage, and putrefying animal matter had long been regarded as a cause of fever,[i] but it was not shown, that the fever thus produced differed from that resulting from other

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[b] GENDRON, 1834, p. 13. [c] PIEDVACHE, 1850, p. 137.
[d] WOOD'S *Pract. of Med.* 4th ed. I. p. 389. [e] JACQUOT, 1858, p. 306.
[f] TROUSSEAU, 1861, p. 179. [g] CHOMEL, 1834. [h] STEWART, 1840, p. 295.
[i] See LASSÔNE, 1749; PRINGLE, 1752, pp. 324-8; *Report of Poor Law Commissioners*, 1842.

causes. In an essay presented to the Royal Medical and Chirurgical Society, in March, 1858,[k] I endeavoured to prove, that fever arising from sewer emanations, was always enteric fever, and never typhus or relapsing fever; and to show that this mode of origin explained why enteric fever was endemic in many places, but often epidemic in circumscribed localities; why it attacked the rich as well as the poor; why it occurred in isolated country-houses, as well as in large towns, and why it was most prevalent in autumn, and in warm seasons. Subsequent observations have tended to confirm the opinions then expressed. The details of a portion of the evidence bearing on the point may now be of service.

In August, 1829, 20 out of 22 boys, at a school at Clapham, within three hours, were seized with fever, vomiting, purging, and excessive prostration. One other boy, aged 3, had been seized with similar symptoms two days before, and died comatose in 23 hours; another boy, aged 5, died in 25 hours; all the rest recovered. Suspicions were entertained that they had been poisoned, and a rigorous investigation ensued. The only cause which could be discovered was, that a drain at the back of the house, which had been choked up for many years, had been opened two days before the first case of illness, cleaned out, and its contents spread over a garden adjoining the boys' play-ground. A most offensive effluvium escaped from the drain, and the boys had watched the workmen cleaning it out. This was considered to be the cause of the disease by Drs. Latham and Chambers, and by others who personally investigated the matter. The morbid appearances in the two fatal cases were described as 'like those of the common 'fevers of this country.' Peyer's patches, and the solitary glands of the small and large intestines, were enlarged like 'condylomatous 'elevations,' and, in one case, the mucous membrane over them was slightly ulcerated. The mesenteric glands were enlarged and congested.[l]

This event is mentioned first, merely from the date of its occurrence. It may fairly be objected, that the course of the disease was more rapid than what ordinarily characterizes enteric fever; but this circumstance was accounted for by the intensity of the poison. Cases of undoubted enteric fever will be alluded to hereafter, in which violent delirium occurred on the first or second

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[k] Murchison, 1858 (No. 1). [l] See *Bibliography*, 1829.

day; and Trousseau records a case which was fatal in less than four days.[m]

In June, 1861, a case, similar to those at Clapham, came under my observation. A girl, aged 9, was admitted into the Middlesex Hospital, under Dr. Stewart, with febrile symptoms, vomiting, purging, and intense headache, followed by acute delirium. She died 47 hours from the first commencement of her illness. The solitary glands in the lower two yards of the ileum, and in the colon, were found enlarged to the size of a hemp-seed or split-pea, and contained a yellowish-white morbid deposit. Peyer's patches were also similarly affected, but there was no ulceration.[n] The mesenteric glands were as large as hazel nuts, and congested. Two or three weeks before, this girl's father had been seized with vomiting and purging, followed by fever, which confined him to bed for three weeks. Accompanied by Dr. Stewart, I visited the rooms, over a stable, occupied by this girl's family. The privy was in the stable, and drained into a cess-pool near the door, which had become choked up. Over the cess-pool was an open grating, by which the stable drained into it, and from which the most offensive smells had issued since the beginning of the warm weather; so offensive, that the horses had sometimes to be removed. The girl had been playing close to this grating at the time of her seizure. The cess-pool did not communicate with the public drain, and no other cases of fever had occurred in the mews.

In 1838, Dr. Ogier Ward recorded the account of a circumscribed outbreak of fever at Birmingham. He proved the identity of the fever with the '*affection typhoïde*' of French writers, by finding ulceration of the ileum and enlargement of the mesenteric glands after death. All the cases, about 50 in number, occurred in the immediate neighbourhood of a small stream, which was nothing more than an open common sewer. The preceding season had been extremely hot, so that the stream was dried up, and at some places almost stagnant and disengaged extremely fetid odours, especially during the night, which were much complained of by the inhabitants.[o]

Towards the end of 1838, an epidemic of enteric fever desolated the commune of Prades in the department of Ariège. Of the 750 inhabitants, 310 were attacked, and 95 died. The cause of the epidemic was traced to a *stagnant* pool, which was the receptacle of

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[m] TROUSSEAU, 1861, p. 168. Boudet records a case fatal on the 6th day, in which *deep ulcers with adherent sloughs* were found in the ileum. BOUDET, 1846.

[n] A figure of the lower end of the ileum will be found under the head of 'Anatomical Lesions.'

[o] WARD, 1838.

dead animals and of all the sewage of the district. The outbreak was preceded by damp warm weather. Three times the pestilence returned, and always when the wind was blowing over the infected water.[p]

A remarkable instance of a circumscribed outbreak of fever was recorded by Dr. Christison, in 1846. It occurred in an isolated farm-house in the thinly-peopled county of Peebles, N.B. Every one of the 15 residents was seized with fever, and 3 died. Many of the servants who worked during the day at the farm were also affected, but none communicated the disease to their families, who did not visit the farm. The only explanation of this outbreak was, that 'the drains and sewers were found all closed 'up, and obstructed with the accumulated filth proceeding from the 'privies and farm-yard,' the effluvia from which were very offensive. Although enteric fever had not, at that time, attracted much attention in Scotland, Dr. Christison observed, with regard to this outbreak, that its 'want of resemblance to the habitudes of ordinary 'epidemic typhus struck the attention as something very remark-'able'; and that 'the leading symptoms were those of great 'gastro-intestinal derangement,' so much so, that suspicions of poisoning were entertained. Moreover, the lengthened duration of the cases, the clearness of the intellect, and the marked absence of prostration, oppression, and delirium, seem to leave little doubt that the fever was enteric. The absence of diarrhœa and abdominal tenderness noted in some of these cases is not incompatible with enteric fever.[q]

About Easter, 1848, a formidable outbreak of fever occurred in the Westminster School and the Abbey Cloisters; and for some days there was a perfect panic in the neighbourhood respecting what was called the 'Westminster Fever.' Within a little more than eleven days it affected thirty-six persons, all of the better class; and in three instances it proved fatal. Shortly before its first appearance, 'there occurred two or three days of peculiarly hot weather,' and a disagreeable stench, so powerful as to induce nausea, was complained of in the houses in question. It was found that the disease followed very exactly in its course the line of a foul and neglected sewer, in which fæcal matter had been accumulating for years without any exit, and which communicated by direct openings with the drains of all the houses in which the fever occurred. The *only* exception was that of several boys who lived

[p] Brichetau, 1841. For other outbreaks of enteric fever in France, traced to similar causes, see *Mém. de l'Acad. de Méd.* ix. 41; xiv. 14; xv. 6.

[q] Christison, 1846.

in a house at a little distance, but who were in the habit of playing every day in a yard, in which there were gully-holes opening into the foul drain. The Metropolitan Sanitary Commission gave it as their decided opinion, that the epidemic 'arose from the bad state 'of the sewers and drains of the precinct, and especially from the 'foul condition of the large sewer described.' Dr. Watson also expressed his belief, that the 'Westminster Fever' was due to the effluvia from this drain; but he did not consider that the cases were Continued Fever at all. Dr. Watson, however, only saw one of the cases; and he expressed the above opinion before he recognized enteric fever as distinct from typhus. Dr. Todd, Dr. Fincham, and Mr. M'Cann, were the other medical men consulted. Dr. Todd, who saw five or six of the cases, informed me that they were unquestionably examples of enteric fever. Dr. Fincham, who, by the way, also saw the case alluded to by Dr. Watson, writes to me, that all the cases which he saw 'were 'unquestionably examples of typhoid fever. In all, the bowel 'complication (the diarrhœa, etc.) was well marked. I believe 'that every case that occurred exhibited the same symptoms.' The same opinion has been expressed to me by Mr. M'Cann. Lastly, two of the cases were admitted into the London Fever Hospital. Their symptoms were recorded, and exactly corresponded with those of enteric fever, including the lenticular rose spots.[r]

Towards the end of the autumn of 1852, a fever broke out at Croydon, which attracted great attention, and was made the subject of various official reports by the Board of Health. Out of a population of about 16,000 persons, 1800 were attacked with fever, and 60 of them died. That this fever was enteric or typhoid, is clearly shown by all the accounts of it which appeared; and also by the expressed opinion of a committee of the Epidemiological Society, consisting of Drs. A. P. Stewart, Jenner, and Sankey. Five cases, moreover, were admitted at this time into the London Fever Hospital, from Croydon: all were enteric. In the month of August, an attempt had been made to drain the town by means of small stoneware pipes, which were not only of insufficient size, but were imperfectly cemented together. Dr. Arnott and Mr. Page, C.E., in their report to the Secretary of State, stated that the escape of sewer miasms had been influential in producing the epidemic. Numerous instances were mentioned, in which the disease immediately followed exposure to the fetid emanations liberated during the opening and emptying of cess-

[r] See *Bibliography*, 1848.

pools, the cleansing of old drains and open ditches, and to the foul gases which were forced into the houses when any obstruction took place in the narrow drain-pipes.[s]

In November, 1853, a remarkable outbreak of enteric fever occurred at Cowbridge, in Wales. Two balls had been held at the hotel of that town, and had been attended by about 140 persons, from all parts of the surrounding country. Shortly after, many of these persons were seized with well-marked enteric fever, and about 8 died. This fever was not prevalent at Cowbridge at the time, and it only attacked those even of the residents who had attended the ball; some were not taken ill until after their return to their homes in Devon and Somerset. At first, suspicions of poisoning were entertained. An inspection of the hotel was made by order of the local authorities. On the day that the inspector made his visit, a very offensive smell proceeded from a privy in the passage leading from the stable to the house. The landlord stated that the smell was due to the privy having been cleaned out that morning, but why it was cleaned immediately before the inspector's visit does not appear. The loft over the stable had been used as a supper-room at the ball.[t]

In the spring of 1857, a number of strangers came to reside at the National Hotel, Washington, to be present at the inauguration of Mr. Buchanan as President of the United States. A large number of them, including the President elect, were seized almost at the same time with enteric fever. It was reported, that they had all been poisoned; at first it was said with arsenic, for some political purpose; and then by copper, from the culinary utensils. A rigorous investigation ensued; and the result was, that both the committee appointed for this purpose and all the medical attendants coincided in the belief, that the disease was due to sewer-gases. At one part of the building there was a direct opening into the sewer, and through this a strong current of fetid air was distinctly perceptible. The fever first appeared after three very warm days, during one of which the rain fell in torrents. The sudden rise of the river Potomac, into which the sewer opened, was thought to have driven back the noxious vapours through the gully-hole.[u]

During 1857, six policemen were admitted into the London Fever Hospital from the Peckham Police-station, with enteric fever: 3 in June, 1 in July, 1 in August, and 1 in September. On inquiry,

[s] See *Bibliography* 1852.

[t] Camps, 1845. This case is here cited, more to show the *local* origin of the disease, than as demonstrating its connection with organic impurities.

[u] See *Bibliography*, 1857.

it was stated, that there was no defect in the drainage of the building, and that the water-closets opened into the drains, and were well trapped. The men, however, affirmed that they had often complained of dreadful odours in the room in which they sat. I accordingly applied to the officer of health for the district, to have the building carefully examined. The result was the discovery that one water-closet on the ground-floor emptied itself, not into the main drain with which it had no connection whatever, but into an old well, immediately underneath the passage adjoining the room in question. Here an accumulation of upwards of ten feet of soil had been going on for years; and the top of the well was merely covered by the flag-stones of the passage. The cess-pool was removed, and the fever ceased.

In the autumn of 1857, enteric fever broke out in Fleet-lane, London, while a sewer was being constructed. The sewer was open from June 29th to October 30th, and during all this time, the inhabitants complained of the offensive smell. Soon after the sewer was opened, diarrhœa began to appear, and enteric fever followed. Of 140 families in the lane, hardly one escaped. Dr. Letheby and Mr. Ross, who investigated the circumstances, both attributed the fever to the sewer miasms. It appeared soon after the sewer was opened, and it disappeared when the sewer was closed, and all the time it was confined to the lane and immediate neighbourhood.[x]

A few years ago, a remarkable outbreak of enteric fever occurred in the Boys' School attached to the Colchester Union, for the particulars of which I am indebted to Mr. Laver. 'Twenty-eight

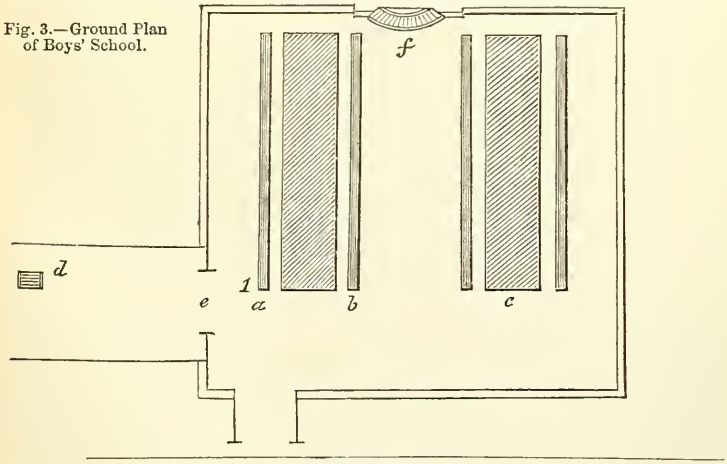

Fig. 3.—Ground Plan of Boys' School.

[x] See Letheby, 1858.

'boys (out of about 36) were attacked; but the first and worse cases 'were those occupying the forms in the school-room marked *a* and '*b*; and, amongst these, the first case was the boy in the 'position marked 1; the cases among the boys at desk *c* 'were very slight. All the boys slept in similar rooms, and 'were similarly treated in other respects.' Mr. Laver had no doubt that the fever was due to gas from an untrapped drain in the passage, marked *d*. He adds: 'You will see that the boys on 'the forms *a* and *b* were in the line of draught between the door *e* 'and the fire *f*, which at that time was burned every day. The 'drain was trapped, and the fever quickly disappeared.' There was no mistake as to the nature of the fever, which 'answered 'well to Dr. Jenner's description of *typhoid* fever.' Every possibility of importation appeared to be excluded, although this point was carefully investigated by Mr. Laver and Dr. Duncan. There were no other cases of fever before or after, in the rest of the Union. 'The boys first attacked had been resident in 'the Union for years, and had not had a day out for a long 'time.' The building was situated out of the town, and the drains of the Union only communicated with those of two or three houses of the best class, in none of which had any cases of fever occurred.

In the autumn of 1858, an epidemic of enteric fever occurred at Windsor, which was made the object of special inquiry by the medical officer of the Privy Council, and an account of which, founded on my own investigations, was communicated to the Epidemiological Society. It was calculated that, during the last four months of the year, 440 persons, or about one-twentieth of the entire population, were attacked, of whom 39 died. The characters of the fever were well marked: such as a red, fissured tongue, abdominal pain, tympanitis, diarrhœa, hæmorrhages, an eruption of lenticular rose spots appearing in successive crops, and a duration of three or four weeks. That the fever was due to the emanations from the sewers was the undisputed opinion of all who investigated the circumstances. Most of the cases, and all but one of the fatal cases, were confined to two of the three districts of the town, the low level and high level districts. Both of these districts had a complete system of drainage, with water-closets within the houses, and sinks in the basements and kitchens. The drains in these two districts were flushed partly by a continuous flow of water through them from the Thames, and partly from artificial tanks. But, in consequence of a long-continued drought, the Thames had fallen greatly in its level, while the

tanks had, from neglect, been allowed to get dry. The result was, that the sewage accumulated in the sewers, and in consequence of their ventilation being very imperfect, the sewer-gases escaped directly into the houses. In the two districts mentioned, the fever attacked the rich and poor indiscriminately; but the cases were most numerous and severe in that part of the low level district, where all the drains of the town converged, and where they had the least inclination, that is, at the foot of Sheet-street near the Barracks.

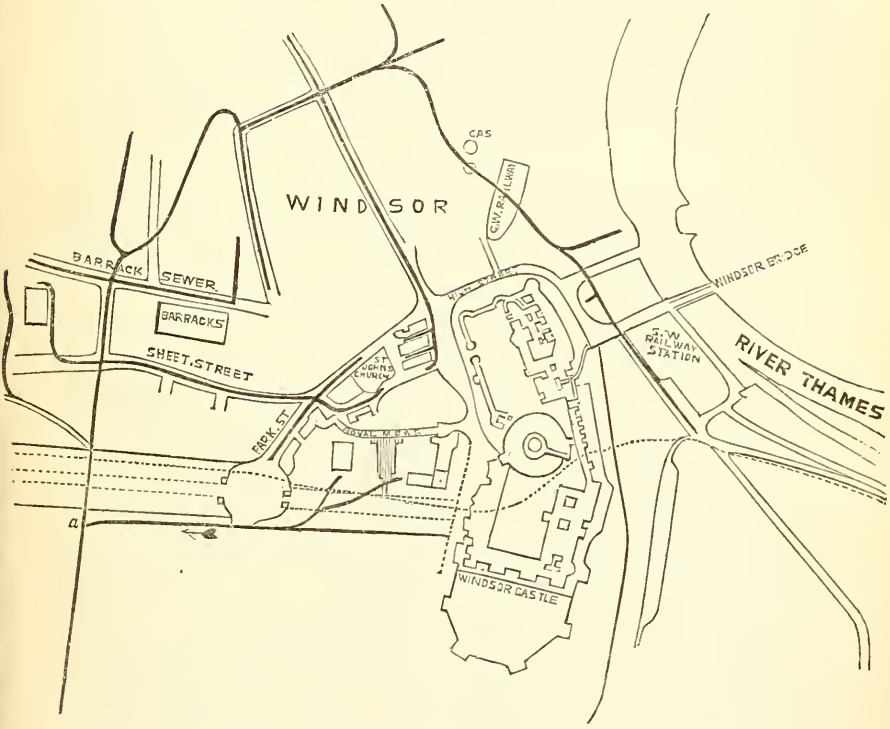

Fig. 4.—Plan of the Windsor Drainage. At *a* the private drain from the Castle joins the main drain outside the town.

The inhabitants in these districts complained of the offensive smells from the drains in their houses, and particularly in the houses where the fever occurred. The district of the town which remained almost exempt from the fever was the worst and poorest, where cholera had raged with greatest severity in 1849. Although the drains of this district also suffered from want of water, the water-closets were almost invariably outside the houses, and there was no direct communication by sinks, or otherwise, between the

drains and the interior of the houses. With few exceptions, bad smells were not complained of in this district. One woman, however, complained bitterly of the offensive smell from the gully opposite her door; her daughter had died of fever. No case of fever occurred in Windsor Castle, which, as may be seen from the annexed wood-cut, had a drain of its own, unconnected with the town drainage. This drain was well ventilated, and was flushed every morning by a special supply of water. A few of the houses in the Royal Mews, connected with the private sewer of the castle, participated in this exemption; but in the remainder of the Mews, only separated by a roadway from the more favoured portion, but connected with the town drainage, there were 30 cases and 3 deaths (see Fig. 4). Lastly, a few cases of fever occurred in the collegiate residences of the castle, which were also connected with the town drainage.[y]

In most of the instances above detailed, the poison appeared to be contained in the volatile emanations from drains, cess-pools, &c.; but, in other cases, it is probably taken into the system through the medium of drinking-water.

In the latter part of 1859, Bedford was the seat of a severe outbreak of enteric fever, although before this it had been 'the 'autumnal habit of the town to suffer from it.' On investigation, it was found, that the distribution of the fever did not follow the ramification of the sewers, nor did it appear to depend on the escape of cess-pool air into the houses: but there was every reason to believe that it was due to fæcal matter soaking into the wells from the numerous cess-pools of the town. The water from these wells was found to contain a large quantity of decaying animal matter, evidently derived from the sources alluded to.[z]

Richmond Terrace, Clifton, is a crescent composed of 34 houses. In 1847, the inhabitants of 13 of these houses drew their drinking-water from a well at one end of the crescent. The remaining houses were supplied with water from another source. At the end of September it became evident, from the taste and smell of the water from the pump, that it was tainted with sewage. Early in October, 'intestinal fever' broke out nearly at once in all the 13 houses, in which the tainted water had been drunk; but did not make its appearance in any of the other houses. In almost every one of the 13 houses, 2 or 3 persons were laid up, and in some a much larger number. The houses, in which the fever broke out, were far apart in the terrace, and there was little or no intercourse

[y] Simon, 1859; Murchison, 1859 (No. 3). [z] Simon, 1860.

between their inmates. The water from the well was the sole connecting link.[a]

In most cases, however, the poison is taken into the system through the air. Many instances might be cited, where, although the water supply was the same to all, only those persons exposed to sewer-emanations have been attacked. For example, in the Windsor epidemic, all the residents in the Royal Mews derived their water from one source: but while 30 cases of fever occurred among those exposed to sewer emanations, not one was observed among those living close at hand, who were exempt from them.

Many other examples of the intimate connection between enteric fever and sewer-emanations, or contaminated drinking-water, have come under my notice during the last five years;[b] but sufficient evidence has been adduced to remove all doubt from most minds on the matter. So close and constant is this connection, that the remark made by several recent French writers with regard to typhus, seems equally applicable here: 'On peut faire naître 'la fièvre typhoïde à volonté, pour ainsi dire.' (See page 110).

In connection with this subject, several experiments on the effects of putrid emanations on the lower animals may be alluded to. Many years ago, Messrs. Gaspard, Magendie,[c] Leuret, and Hammond,[d] showed that, by injecting putrid substances into the veins of animals, symptoms very similar to those of enteric fever might be induced, and that after death the intestines were much congested. The same results have been obtained by D'Arcet,[e] on injecting into the veins putrid pus. Magendie also made experiments on the effects of inhaling the gases emitted by putrefying animal substances. Into the bottom of a cask he introduced putrid substances, and in the upper part he placed an animal, supported on a second grated bottom, so as to expose it freely to the emanations from below. In one dog, which died on the tenth day, the intestines were found much inflamed. Although none of these experimenters succeeded in producing the specific lesions of enteric fever, the putrid substances which they employed differed from that which probably produces the poison of this disease. So far as we know, this poison is chiefly produced by fæcal fermentation. In 1858, Dr. Barker of Bedford, published the results of some interesting experiments, which consisted in making animals inhale

[a] W. Budd, 1859, p. 432. For other examples, see Routh, 1856, p. 763; Mauer, 1862; and Fever at Munich, *Bibliog.* 1862.

[b] See also Murchison, 1858 (1).

[c] Magendie, 1823.

[d] Leuret and Hammond, 1827.

[e] D'Arcet, 1842.

cess-pool gases.[f] The animals were placed in a closed chamber, through which a constant current of cess-pool air was kept up. In most of the animals, vomiting and purging were produced, and in one, where the experiment was prolonged, the symptoms were not unlike those of enteric fever; but no mention is made of the *post-mortem* appearances in any case. Dr. Richardson, however, states, that he has succeeded in producing 'patches of ulceration 'along the alimentary tract' of a dog, by making it inhale sulphide of ammonium, one of the gases given off by cess-pools.[g] Further investigations in this direction are very desirable.

It is necessary now to notice certain objections to the origin of pythogenic fever in the manner above described.[h]

1. The most important is that of which Dr. W. Budd is the chief and able exponent.[i] According to Dr. Budd, the poison of enteric fever, although contained in sewage, is then always derived from the alvine evacuations of an individual already suffering from the disease; the poison resides chiefly in the stools of the sick, and a drain is merely the vehicle of its propagation, or, in fact, 'a direct continuation of the diseased intestine.' Admitting fully, that the view advocated by Dr. Budd and Professor von Gietl,[k] offers the best explanation of the circumstances in those cases where the fever is propagated by the sick, many, if not most, of the facts adduced in favour of it are explicable on the theory of spontaneous generation; while in others, the mode of communication is not so clearly established as might be desired. Dr. Budd's arguments are two-fold: first, he adduces many facts to show that the disease is contagious; and secondly, he mentions many cases to demonstrate the intimate connection between its origin and bad drainage. Both of these positions I readily concede, and have always contended for. But it does not appear to me equally clear, that, in the cases recorded by Dr. Budd, the disease was propagated by the stools of persons previously infected. For example, in the North Tawton Fever, on which so much stress has been laid, while the facts leave little doubt that the fever was communicated in some instances by the sick to persons in health, it is not shown that the stools of the infected were the medium of communication.[l] On the other hand, Dr. Budd records 3 instances, from which he argues that sewers merely transmit the poison, in consequence of receiving the excreta of a

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[f] Barker, 1858. [g] Richardson, 1858, p. 345. [h] See also page 8.
[i] Budd, 1856, 1859, 1861. [k] Gietl, 1860.
[l] Budd, 1856, p. 695; 1859, p. 29.

diseased intestine.[m] In all of these instances, the fever evidently arose from air or water tainted with sewage; but it is not shown that the sewage in any of the cases had first become contaminated with the excreta of a person suffering from enteric fever.[n] The necessary link in the evidence, viz., the introduction of the poison, is wanting. In another instance, cited by Dr. Budd, where 4 cases of fever occurred in a retail establishment at Bristol, it was argued that the disease in the last 3 cases was due to the evacuations of the first case being thrown into the common water-closet. But it was not shown that the poison was imported by the first case, which is spoken of as 'casual,' and on the supposition that the first case was due to some local cause, that cause was sufficient to account for all.[o] In other instances, such as the Orphan Asylum at Ashley Hill, and the school in the South of England, where the fever appeared in connection with offensive latrines, it is also argued that the cases were due to the children frequenting the latrines into which the dejections of the first patient had been thrown, but it does not appear that the first patient contracted the disease elsewhere than in the asylum or school.[p] Even in the account of the North Tawton outbreak, although the date and locality of the first case are mentioned, it is not stated that the patient caught the disease away from the place. The circumstance much dwelt on by Dr. Budd, that extensive outbreaks of enteric fever have occasionally been preceded by two or three isolated cases, proves nothing in favour of contagion, in my opinion, except it can be shown that in these first cases the fever was contracted away from the site of the subsequent outbreak. Moreover, in not a few instances, many persons have been attacked simultaneously in the same house, without any isolated cases preceding.

But, on the supposition that enteric fever is occasionally propagated in the manner described by Dr. Budd, it does not necessarily follow that, in every case arising from bad drainage, the poison has been merely transmitted.[q] On the contrary, there are facts, which, in my opinion, demonstrate that the fever often arises

[m] W. Budd, 1859, pp. 432, 458; 1861, p. 550.

[n] In one of the instances, it is stated, that *a few days* before the water of a certain well was *discovered* to be contaminated with sewage, there was a single case of fever in an adjoining house. But it is not shown, that this patient contracted the disease elsewhere than in the house in question, or that diarrhœa had occurred, before the patients in the next house began to be ill. Dr. Budd remarks: 'Whether or not this case was the source of the specific 'poison, from which the others sprang, we need not inquire.' It appears to me, that he has omitted to place the key-stone in the arch of his argument.

[o] Budd, 1856, p. 618; 1859, p. 458. [p] Ibid,

[q] On this subject, the reader is referred to page 8.

independently of any such transmission. It is difficult to obtain crucial evidence on the point from what is observed in large towns, furnished with a complete system of drainage, because, to every instance of fever arising from the inhalation of sewer-gases, or from drinking water polluted with sewage, it would be replied, that sewer emanations are 'the very quintessence of a pre-existing 'fever.' But even in towns, evidence is not wanting, that enteric fever has arisen from bad drainage, where it is impossible to conceive that the poison was introduced into the drain. For example, in the case of the fever at the Peckham Police Station (see page 442), the cess-pool had no communication with the public drains; in the outbreaks at Westminster (see page 440), Clapham (see page 438), and other places, the source of the fever was traced to the decomposition of sewage in drains which were *choked up*, and so, in a measure, shut off from the general drainage; while in the case of the Colchester Union(p. 443), every possibility of importation appears to me to have been excluded. On turning to what occurs in country-districts, the evidence is still more conclusive. Several instances have come under my notice, in which enteric fever has broken out in an isolated country-house, or in a small group of houses, miles away from where any fever was prevailing, and in which every mode of importation, or of communication by drains, or otherwise, seemed impossible; and by numerous inquiries from country practitioners, I learn that such events are far from rare.[r]

There is another view of the subject worth considering. Dr. Budd, like many former writers, regards the intestinal disease as a specific eruption, and he contends that an 'infinitesimally small 'dose of the poison' derived from this eruption, is sufficient to produce the disease.[s] But there is no positive evidence that the stools of enteric fever are of such a virulent character, while there are some circumstances which rather tend to an opposite conclusion. As already stated, since my connexion with the Fever Hospital, 1,048 cases of enteric fever have been under treatment, but only one case of the fever has originated in the hospital, and yet the night-stools containing the excretion often remain for many hours without being emptied. Moreover, on several occa-

[r] Several remarkable instances of this nature, in which the circumstances were most carefully investigated, were communicated to the Epidemiological Society, by Dr. Headlam Greenhow, on April 7th, 1862. Dr. G. remarked: 'No discoverable evidence of its having originated in contagion, could be 'traced on the most careful inquiry.'

[s] Budd, 1859, p.209.

sions, I have known other patients sit over the evacuations of enteric cases, without suffering. Surely, if these stools are so venomous, as Dr. Budd would have us believe, a contrary result might have been expected. Some years ago, I fed a pig for six weeks on the stools of patients suffering from enteric fever. They were mixed with barley-meal, and given two or three times a day. The animal appeared to suffer no inconvenience, but, on the contrary, it got very fat, and when killed, its intestines were perfectly healthy.[t] Again, if the stools of enteric fever are so virulent, that those of one patient can give the disease to a whole community, as Dr. Budd believes, an epidemic might be expected to be constantly raging on the banks of the Thames, which must often contain the excreta of many thousands of enteric patients; yet such is not the case.

A careful examination of the stools in enteric fever, shows that they are remarkably prone to decomposition, or fermentation. In place of being acid, as healthy fæces always are, they are invariably alkaline; they also contain abundance of the ordinary products of the decomposition of animal matter, in the form of ammonia, and ammoniaco-magnesian phosphate, while their odour fully bears out the idea of their putrid character. Surely such a condition would be fatal to the existence of an animal poison, such as that contained in the pus of small-pox. Dr. Budd says, that the morbid material deposited in the intestines contains the specific poison by which the disease is propagated, just as the contents of the variolous pustule contain the poison of small-pox. But if this be so, the poisonous matter is never passed from the body until it separates *as a slough* from the intestine, *until, in fact, it is dead and putrid.* It has yet to be shown, that the contents of the variolous or vaccine pustule, will produce small-pox or cow-pox, after they become putrid. Lastly, the fact that the prevalence of the disease is influenced by *temperature* (see pages 416, 419), is opposed to the idea that it depends on a specific poison derived from the sick, but is readily accounted for, on the supposition that the poison is generated by fermentation or decomposition.

It is very probable, that the stools of patients, suffering from

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[t] For the details of the experiment, see MURCHISON, 1858 (No. 4).

The lower animals are not exempt from enteric fever, as has been alleged. Professor Gamgee of the Veterinary College of Edinburgh, has informed me, that a disease, corresponding to it, in its symptoms and anatomical lesions, is well known to veterinary pathologists, to exist in pigs, horses, and other animals, and he has furnished me with references to numerous memoirs, in which it has been described. See especially, FALKE, 1840.

enteric fever, are more prone than ordinary sewage to the peculiar fermentation by which the poison is produced, and even that, in certain cases, the fermentation may have commenced before their discharge from the bowels. In this way, enteric fever may occasionally be propagated by the stools. But whether this be so or not, it seems to me far more probable, that the poison is *always* the result of *decomposition*, than that it is derived from a *specific eruption*, like that of small-pox. While then, I entirely agree with Dr. Budd in recommending the immediate removal of the discharges from the sick, I hold that it is even more necessary to rectify without delay the escape of sewer-gases into houses, and the pollution with sewage of drinking water.

2. The second objection to the theory of spontaneous generation now advocated, is, that persons are often exposed for a long time to the emanations from decomposing animal matter, without contracting enteric fever. Numerous instances of this sort have been brought forward, with the object of making the doctrine appear ridiculous. Many of the examples adduced, however, have no bearing on the question. Because fæcal fermentation generates the poison of enteric fever, it does not follow that this poison is contained in the exhalations from every form of animal matter undergoing decomposition, such as from dead bodies during exhumations, or in a dissecting-room, from old bones, from putrid blood employed for refining sugar,[w] or from the horse-slaying yards of Montfauçon.[x]

Again, it is not to be supposed, that every heap of rubbish, or uncovered dung-hill, however offensive, can generate the poison of enteric fever, or that this poison is contained in every bad smell. The poison itself may be inodorous, although, in most cases, it is associated with fetid gases. It must be remembered, that the miasmata which give rise to intermittent fever, are inappreciable by the senses, or by chemical research. So far as we know, it is necessary for the production of the poison of enteric fever, that the matter undergoing fermentation be either in a confined space, as in a drain or sewer, or that it be in a state of stagnation. Free exposure to the atmosphere, or constant dilution in a running stream, may not only render the poison inoperative, but may altogether prevent its formation. Thus we account for the circumstance, that no extensive ontbreak of fever occurred in London in

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[w] Chisholm, 1810; Bancroft, 1811, p. 634; R. Williams, 1836.
[x] Du Chatflet, 1832.

1858, in connection with the unusually filthy and offensive condition of the Thames.[y]

Moreover, in most of the instances where enteric fever has been traced to bad drainage, the noxious gases have escaped into the interior of the houses. Hence, as was notoriously the case at Windsor, in 1858; a privy outside a house is much less dangerous than a badly appointed water-closet within. (See page 445).

In the next place, it has been urged, that the workers in drains and night-men are particularly exempt from fever. The facts chiefly appealed to in support of this statement are those recorded by Parent Du Chatelet[z] and Dr. Guy.[a] But a careful consideration of the facts in question does not justify such an inference. In an Appendix to Du Chatelet's Essay on the Diseases of the Workmen in the Drains of Paris, it is stated that, during six months, 4 of the 32 workmen, who formed the subject of the essay, were in hospital for two or three weeks with a '*fièvre bilieuse*,' or a '*fièvre bilieuse et cérébrale*.' Although these cases are not dwelt on in the body of the essay, it is impossible to regard them in any other light than as examples of enteric fever. Again, the result obtained by Dr. Guy was simply this: that whereas of 101 labourers and brickmakers 32 had suffered from fever; of 96 nightmen, scavengers and dustmen, only 8 had suffered. Dr. Guy's observations, however, were made without any reference to the form of fever, in either case, and, indeed, at a time when the distinctions between the different continued fevers were little known; but still there is evidence that the excess of fever among the bricklayers' labourers was *typhus*, inasmuch as it was attributed to the men being Irish, and to their habits of over-crowding. Moreover, there is no reason to believe that scavengers or dustmen are unusually exposed to sewer-emanations. Dr. Guy fully admitted that certain cases of fever were generated by the emanations from drains and cess-pools. According to Dr. Peacock's experience, enteric fever is not uncommon among the workers in sewers;[b] and several cases which have come under my own notice, lead to the same conclusion (see page 426). The disease would probably be more common in this class of labourers, were it not that most

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[y] The oft-repeated argument, that, during this year, the prevalence of *fever* in London was much below the average, requires a word of explanation. The fact was owing to the almost total disappearance of *typhus* (see pages 52, 64), which, in the two former years, had been so prevalent. There was no decrease of *enteric fever*, as I ascertained by numerous enquiries at the time, and as is shown by the returns of the Fever Hospital (see pages 413, 417).

[z] PARENT DU CHATELET, 1829. [a] GUY, 1848.

[b] PEACOCK, 1856 (1).

of them are above the age most liable to enteric fever, that some are protected by previous attacks, and still more, that lengthened exposure to the exciting cause diminishes the risk of infection. Whatever be the cause of enteric fever, it is generally admitted that constant exposure fortifies the system against its action (see page 425). In the outbreak at Clapham, already alluded to, although 20 out of the 22 boys suffered from the opening of a drain, the workmen, who went down into the drain, escaped. Hence, on the supposition that sewer-emanations can produce enteric fever, it is not so surprising, as it might at first seem, that persons, most exposed to them, suffer less than others, whose exposure is casual.

Lastly, it is probable, that a certain temperature (see pages 416 and 419), and, perhaps, other atmospheric conditions, are necessary, for the production of the poison of enteric fever during fæcal fermentation, and also that the poison is only generated at a certain stage of the fermenting process. Nuisances may appear to exist unchanged for many years, although the emanations proceeding from them vary greatly at different times.

3. Thirdly, it has been argued, that many cases of fever are independent of organic impurities; but this objection has mainly arisen from all forms of continual fever being regarded as one disease. Twenty years ago, a memorable discussion took place between the late Dr. Alison, of Edinburgh,[c] and the London Poor-Law Commissioners.[d] The London observers showed that 'fever' often arose from putrid emanations, and was independent of destitution, whereas Dr. Alison brought forward abundant evidence to prove that destitution was the chief cause of its propagation, and that putrid emanations had nothing to do with it. Both were right, but their observations were made on different diseases.

At the same time, I readily admit, that we cannot succeed in tracing every case of enteric fever to organic impurities. *But if the disease can be traced to such causes, in a few undoubted instances, it is reasonable to infer, that its causes are similar, in all cases where it has a spontaneous origin.* As already stated, *the actual poison may, like the miasmata which give rise to ague, be inappreciable by the senses, or by chemical research.* During the last four years, however, I have met with few examples of enteric fever, which, on investigation, I could not trace to defective drain-

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[c] ALISON, 1840 (No. 2); also PERRY, 1844, p. 84.
[d] ARNOTT, 1840; *Report*, 1842.

age, the existence of which was occasionally unknown to the inhabitants of the infected locality.

One other circumstance must be referred to, in connection with this subject. It is probable that the poisons of other diseases besides enteric fever, may be derived from fæcal fermentation. For example, circumscribed epidemics, or the ordinary autumnal increase of enteric fever, are often preceded by a great increase of diarrhœa; and it is worth noticing, that the diarrhœa reaches its *acme* long before the fever does, and that by the time the latter is most prevalent, the former has greatly declined. This was observed to be the case at Windsor, in 1858,[e] and, as seen by the annexed table, at London, in 1857.

TABLE XXXVI.[f]

Months.	Diarrhœa.		Fever.		
	Cases of Diarrhœa reported to General Board of Health.	Deaths from Diarrhœa reported by Registrar-General.	Total cases of Continued Fevers reported to General Board of Health.	Cases of Pythogenic Fever only, admitted into London Fever Hospital.	Cases of Typhus admitted into London Fever Hospital.
May	633	47	548	1	42
June	1,770	114	704	9	18
July	13,506*	609*	874*	19	35
August	19,557	915	839	26	16
September ...	8,432	519	891	34	14
October	2,846*	232*	1,179*	38	10
November ...	1,118	85	919	33	1
December ...	767	73*	664	29	2
Total.....	48,629	2,594	6,618	189	138

The etiology of pythogenic fever may be thus summed up :—

1. Pythogenic fever is either an endemic disease, or its epidemics are circumscribed.

2. It is most prevalent in autumn and after hot weather.

3. It is independent of overcrowding, and attacks the rich and poor indiscriminately.

4. It is often generated spontaneously by fæcal fermentation.

5. It is occasionally communicated by the sick to persons in health.

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[e] MURCHISON, 1859 (No. 3), p. 305.

[f] The numbers marked * in the first three columns, represent the cases which occurred during five weeks, the others only those for four. This arises from the data being derived from *weekly* returns. The last two columns show the numbers admitted from the first day of one month to the first of the subsequent one. The third column, of course, includes typhus cases; but the fifth shows that this fever was greatly on the decline at the period of greatest prevalence, indicated in the third and fourth columns. The fourth column contains *only* cases of pythogenic fever.

6. This transmission is probably effected by the stools, which are unusually prone to decomposition.

Section VI. Symptoms of Pythogenic Fever.

A. Clinical Description.

THE advent of pythogenic fever is, in most cases, gradual. The patient is often unable to state the precise day on which his illness commenced, although his mind may be clear and his memory good. In some cases, the first symptoms complained of are irregular chills, loss of appetite, headache, pains in the limbs, giddiness, and ringing noises in the ears, with, or without, diarrhœa and sickness. In many cases, relaxation of the bowels, with or without sickness and abdominal pain, is the first symptom which attracts attention, and the patient is thought to be suffering from an attack of ordinary diarrhœa or of gastric derangement. Occasionally, urgent diarrhœa supervenes on the injudicious administration of a purgative. In addition to these symptoms, the pulse is accelerated; the skin is hot; the tongue is furred, and red at the margin; and there may be slight epistaxis. At the same time, the patient has disturbed and restless nights, and he feels weak and disinclined for bodily or mental exertion. Still, he is rarely so prostrate as to keep his bed during the first five or six days of the disease; and it is not uncommon for patients to continue at their ordinary employment, for the first week or ten days, or even to walk to hospital, at the end of a fortnight. The above symptoms manifest themselves in varying degrees of severity during the first week. As a rule, the fever is greatest towards evening, and in the night. As yet, there is nothing absolutely pathognomonic of pythogenic fever; but the concurrence of diarrhœa or gastric disturbance with fever and prostration, in a young person, ought always to make the practitioner suspect, that this is the disease which he has to combat.

At the end of the first week, before which the patient seldom comes under the observation of the physician, the symptoms are as follows:—The pulse is between 100 and 120, but its frequency varies in the same patient. At one time, it may be 90 or 100, and at another 120. As a rule, it becomes accelerated towards evening and in the night, and falls in the morning. Although weaker than natural, it may still exhibit considerable resistance. The skin is warm and dry; but, frequently, and particularly in the morning, it is clammy or moist; its temperature is also lower in the morning than at night. The lips are parched and dry.

The tongue is covered with a thin white fur; its margin and tip are red. The patient has no appetite, but complains of thirst, and has occasionally bilious vomiting. In most cases, the bowels are relaxed; there are two, four, six, or more watery motions, of an ochrey-yellow colour, in the twenty-four hours. When there is no diarrhœa at this time, it is often found to have existed, before the patient applied for advice. The abdomen is distended and tympanitic, and there is often gurgling, on pressure, in the right iliac region. The spleen is enlarged. The urine is scanty, dense, and high-coloured. The headache, giddiness, and disturbed sleep continue; but the mind is usually clear, and the memory unimpaired; there is rarely any delirium even at night. The patient does not exhibit that heavy, stupid expression, so characteristic of typhus; the conjunctivæ are not injected, and there is no general dusky flush of the face; but, on one, or both cheeks, there is often a circumscribed pink flush, not unlike the hectic flush of phthisis, which varies in intensity at different times, but is usually most developed towards evening and after food.

On the seventh day, or some time between this and the twelfth day, an eruption appears on the chest, abdomen, and back, which consists of isolated, small, circular, tolerably well-defined, rose-coloured spots, slightly elevated above the skin, and disappearing on pressure, but returning when the pressure is removed. Their number varies from three or four, to many hundreds; but in most cases, does not exceed twenty or thirty at one time. They are developed in successive crops (See Plates III. IV. and V., and page 469).

About the middle of the second week, the headache and general pains abate; and by the end of the week, they are rarely complained of. About the time that the headache ceases, more or less somnolence supervenes, which is often interrupted by delirium. At first the delirium is slight, and only observed during the night; but gradually it becomes more severe and constant. It varies greatly in its intensity and character in different cases; but, as a rule, it is more acute and noisy than in typhus. In the intervals of delirium, the patient may be perfectly rational and conscious; at other times, he is more or less confused: or occasionally, he lies motionless in bed, with his eyelids closed, but takes notice and appears to understand perfectly what is said to him; he puts out his tongue, and does what he is told at once; and his expression is almost natural; but when spoken to, his answers, although prompt, are inarticulate and unintelligible. At the same time, the pulse is 120, or upwards, and weak; the pupils are dilated; epistaxis may occur; the lips are parched and cracked; sordes collect

on the teeth; the tongue is dry and brown at the base and along the centre, or it is red, glazed, and marked by deep fissures; the abdomen is more distended; the bowels are still relaxed, and the stools contain membranous flakes, and occasionally blood. The urine is more copious, paler, and less dense. Fresh spots continue to appear; but the old ones fade, and are never converted into petechiæ. Bed-sores form over the sacrum and other parts of the body subjected to pressure.

Day by day, the patient loses flesh and strength, and becomes more unconscious, and all the phenomena of the typhoid stage of typhus,[g] viz: dry brown tongue, feeble pulse, low muttering delirium, stupor, tremors, subsultus and involuntary evacuations—may be developed. In this state, death may take place by coma; or gradual improvement may occur towards the end of the third or fourth week of the disease.

But, in a large proportion of cases, the typhoid stage is never developed; while in many there is little or no delirium, and the mind is perfectly clear throughout the attack. Even in cases of this nature, however, the patient is not exempt from danger. Apart from pulmonary and other complications, which may supervene at any period and prove fatal, there are certain risks arising from the local lesions, with which the fever is invariably accompanied. Death may occur at any time during the third and fourth week, from peritonitis consequent on perforation of the intestine, from profuse and exhausting diarrhœa, or from hæmorrhage from the bowels; and then the consciousness may remain unimpaired until the last.

When the disease terminates favourably, the amendment is, in most cases, gradual. It is rarely possible to determine, as in typhus and relapsing fever, the precise day on which convalescence commences. The usual duration of the disease, however, is from three to four weeks. The emaciated condition of the patient renders convalescence at all times tedious, while it is apt to be interrupted by various complications. Occasionally, after convalescence has advanced for ten days or a fortnight, there is a relapse of the original fever, lasting for ten or twelve days, and characterized by a fresh eruption of lenticular rose-spots, and by fresh deposit in the intestinal and mesenteric glands. It follows, that an attack of pythogenic fever is sometimes a disease of two or three months' duration.

Such is the clinical history of ordinary cases of pythogenic

[g] See page 173.

fever. But the picture is susceptible of numerous modifications. There is no disease, in fact, which exhibits a more protean character, from the predominance of certain symptoms, and from the presence of complications. Some cases run a very mild course, so that the patient is scarcely confined to bed; some are at first severe, and afterwards put on a mild character; others, at first mild, undergo a sudden aggravation; while others are severe from first to last. Occasionally, acute delirium is observed almost from the commencement; while, in many cases, there are no cerebral symptoms throughout the disease. Vomiting may be a distressing symptom; and diarrhœa may be profuse and constant; or, on the other hand, there may be no symptoms of gastric derangement, and the bowels may be constipated from the beginning to the end.

B. Illustrative Cases.

Case XXIV.

Pythogenic Fever of moderate severity.—Convalescence at the end of the Fourth Week.

Emma B——, aged 16, servant in a private family, was admitted into the Fever Hospital on October 17, 1858. She stated that her master and mistress, and their son, had all died of the same fever, and that the smell from the closets and sinks of the house were frightful. She had been ill for ten days before admission, but had only kept her bed for two days. Her principal symptoms had been headache, diarrhœa, loss of appetite, and thirst.

Oct 18 (twelfth day). Pulse 96. Expression languid, but not at all stupid. Headache has ceased, but had a restless night. Intelligence is now clear, or is but very slightly confused. Pupils dilated. Skin hot and dry. Two lenticular rose-spots on abdomen. Tongue moist and furred, red at edges; abdomen distended and tympanitic; tenderness and gurgling in right iliac fossa; one watery stool. Was ordered milk, beef-tea, and mistura camphoræ.

October 20 (fourteenth day). Pulse 120. A circumscribed pink flush on left cheek. Has much pain in abdomen, and has passed six copious watery stools of an ochrey colour, in last twenty-four hours. Was ordered acetate of lead (gr. iij.) every four hours.

October 21 (fifteenth day). Is more prostrate. Pulse 104, and weaker. Moans very much in her sleep, but has no delirium, and intelligence seems clear, although she drawls out her replies, and her expression is very listless and languid. Pupils much dilated; slight deafness. Left cheek much flushed; several fresh spots; skin hot and dry, but nurse says she perspires always in the night. Tongue moist and furred; much tenderness in right iliac fossa; six stools, but less copious. Was ordered a starch and laudanum enema, and six ounces of wine.

October 28. Has continued in the same languid state for the last week. Has had no delirium, and, although for the last day or two she has been somewhat drowsy, she always replies when spoken to in the same drawling way as before. The pupils have been always dilated. A few fresh spots have appeared almost daily, and the perspiration has returned almost every night, although the skin has been hot and dry in the day-time. Has only had two stools in last 24 hours, but this morning she vomited about two ounces of green bilious fluid, and abdomen is more distended. Was ordered a turpentine-stupe to the abdomen, and a starch and laudanum enema.

October 30 (twenty-fourth day). Pulse 88. Is very weak, but feels and looks better; intelligence clear, and she answers more quickly; pupils still large. The spots have almost disappeared, and there has been no stool since yesterday morning. Was ordered a mixture containing carbonate of ammonia.

November 1 (twenty-sixth day). Pulse 112, but continues to improve. Complains of pains in back and limbs. No spots visible, and only one stool since October 30. Appetite returning. Was ordered a lightly boiled egg.

November 3 (twenty-eighth day). Pulse 100. One fresh spot has appeared to-day. No motion for two days, and is very hungry; but abdomen is more distended. Wine to be reduced to four ounces; to have two eggs, and a mixture containing quinine and mineral acid.

November 6 (thirty-first day). Pulse 96. Spot marked on 3rd continues, but there are no fresh ones. Two stools yesterday, and one to-day, more watery. Has still much flushing of the face.

November 9. Pulse 92. Tongue moist and clean; one natural stool; no spots visible. Was ordered fish. From this date, the patient improved daily, but she was much emaciated, and was long in regaining her flesh and strength. On November 20, she got up, and on December 11, she was discharged from the Hospital.

Case XXV.

Pythogenic Fever, of a very mild form.—Bowels constipated throughout.—Lenticular Spots preceded by a Scarlet Rash.

Margaret M——, aged 10, was admitted into the London Fever Hospital, August 9, 1858, having been complaining for a week, of headache, giddiness, loss of appetite, thirst, and general debility. Her aunt was laid up at home with fever, and her sister had been admitted into the Hospital ten days before, with all the symptoms of pythogenic fever, including successive crops of lenticular spots and diarrhœa, with ochrey stools.

August 10 (ninth day). Pulse 108 in the morning, and 120 late in the afternoon. Is now free from headache, and intelligence is quite clear Disturbed sleep. Skin hot and dry. Temperature under tongue 103° Fahr. No spots, but has a circumscribed pink flush on both cheeks,

and the whole of chest and abdomen is covered with a bright scarlet rash, like that of scarlet fever. Tongue moist and furred, very red at edges. No sore-throat. Abdomen distended, and tympanitic; there is gurgling in right iliac fossa; but the bowels have not been opened for two days. Was ordered milk and beef-tea, mistura camphoræ, and a teaspoonful of castor-oil.

August 13 (twelfth day). Pulse 96. General condition is the same as on admission; but the scarlet rash has disappeared, and to-day there is a distinct lenticular rose-spot on the abdomen. The bowels have not been opened since admission, except after oil.

August 17 (sixteenth day). Pulse 100. Is free from pain. Intelligence clear, and sleeps well. Expression natural, or only a little languid; pupils much dilated. A deep pink flush on both cheeks. Temperature under tongue 103° Fahr. Perspires at night. Several fresh lenticular spots have appeared every day since 13th, while the one marked on 13th is no longer visible. Tongue moist and clean, but red at edges. Bowels never moved without oil. Respirations 18; has no cough.

August 19 (eighteenth day). Pulse 84. Feels much better. Pupils still large. Temperature under tongue 101° Fahr. Spots have all disappeared, and there is less flushing. Is very hungry.

On August 23, the pulse had fallen to 60, and the temperature was 99°; on the 24th, the pulse was 84; and on the 26th, it was 96. Notwithstanding these variations in the pulse, the patient slowly improved; on the 27th, she was able to leave her bed; and on September 14, she was discharged from the Hospital, well.

Case XXVI.

Pythogenic Fever, of a severe form, lasting upwards of Four Weeks, with Delirium and Fatuity during Convalescence.

John M——, aged 16, was admitted into the London Fever Hospital, September 10, 1858, having suffered for a fortnight from febrile symptoms, headache, and diarrhœa. For four days before admission, he had been very delirious.

September 11 (fifteenth day). Pulse 96, and compressible. Incessant delirium. Talks incoherently about money and railways, laughs at one time and swears at another. Is with difficulty kept in bed. Looks steadfastly at any one who speaks to him, and then bursts out laughing. Pupils natural; countenance pale. Five or six lenticular spots on abdomen. Lips parched; tongue red and dry. Abdomen distended. Five light-yellow watery stools since admission. Was ordered beef-tea and milk; four ounces of wine; acetate of lead (gr. iij,) after each motion; and a starch and laudanum enema.

September 12 (sixteenth day). Pulse 120. Temperature in axilla 105° Fahr. Several fresh spots. Tongue brown, dry, and fissured. Purging quite ceased after second dose of lead.

September 16 (twentieth day). Pulse 112. Is much more prostrate; hands and tongue tremulous. Noisy delirium has continued ever since admission, except when sleep has been procured for an hour or two, by means of an opiate draught. Pupils are small. Several fresh spots have appeared daily, and many of those marked since admission are no longer visible. Is now sweating profusely, and abdomen and chest are covered with sudamina. Lips are bleeding from being picked; sordes on teeth; tongue dry and brown; intense thirst; much tenderness of abdomen. The diarrhœa returned on the 14th, but has again ceased, after a repetition of the lead. Slight epistaxis. Temperature in axilla 104° Fahr. Urine 20½ ounces, containing 292 grains of urea. Six ounces of brandy substituted for wine.

September 18 (twenty-second day). Yesterday pulse was 144; to-day it is 112, and weak; but the cardiac impulse is strong and thumping. Is still talkative at times, but is, on the whole, much quieter, and is drowsy, and almost unconscious; pupils small. Has had no opium for two days. Still perspiring, but skin is at other times dry. Fresh spots continue to appear. Tongue dry, red, and fissured. Much retching, but brings up nothing. Great tympanitis, and much tenderness of abdomen. One stool, viscid, like bird-lime, and extremely offensive. Temperature 102°. Urine thirty ounces, alkaline, and containing 367 grains of urea. Was ordered ten ounces of brandy, and a mixture containing ammonia and chloric ether.

September 20 (twenty-fourth day). Much worse. Pulse 108. Is quite unconscious, and very drowsy, with occasional low muttering delirium. Pupils contracted. Much tremor, subsultus, and carphology; stools and urine passed in bed. Bowels more relaxed.

September 22. In the same state. Occasional flushes on cheeks. Skin over sacrum is red.

September 23 (twenty-seventh day). Marked improvement. Pulse 98. Takes more notice, and is less drowsy, but at times shouts and talks nonsense. Subsultus and carphology ceased, and has less tremors. Pupils natural. Spots fading, and no fresh ones. Tongue moist and very red. Still retches much, but brings up nothing. Abdomen tender. One stool, not in bed. Temperature 100° Urine 31¼ ounces, containing 503 grains of urea.

September 25 (twenty-ninth day). Is worse again. Pulse 96. Much tremor, and is quite unconscious. Spots have all disappeared. Temperature 100¾°. Urine 28 ounces,[h] containing 368 grains of urea.

September 28 (thirty-second day). Pulse 92. Is much better again. Quite sensible when spoken to. Slept at intervals during the night. Pupils large. No spots. Tongue red and moist; four relaxed stools. Temperature 100½°. Urine 42 ounces, containing 638 grains of urea. Small bed-sore over sacrum.

[h] About 4 ounces more of urine were passed with motion.

On September 30, the patient passed 75½ ounces of urine, containing 964 grains of urea, and on October 2, 59½ ounces, containing 703 grains of urea. From September 28, he gradually improved, but for a fortnight he was very talkative at times, and silly in his remarks. All this time, the pulse varied from 84 to 120. The pupils were large; the appetite voracious; and the patient complained bitterly of pains in his joints and limbs. On October 17, he was able to get up; and on October 29, he was discharged from the Hospital.

CASE XXVII.

Pythogenic Fever.—Acute Delirium followed by Coma and Death on the 6th day.—No Diarrhœa.—Autopsy:—Extensive Deposit in Peyer's Patches, but no Ulceration.—Enlargement of Mesenteric Glands.

Arthur F——, aged 6, was admitted into the London Fever Hospital, August 20, 1856. His brother had been admitted on August 8, with pythogenic fever, which was characterized by numerous lenticular spots, and profuse diarrhœa. Arthur F—— had only been taken ill on August 17. His symptoms, before admission, had been great thirst, severe headache, and restlessness; during the two nights preceding admission he had been delirious, but the bowels had not been relaxed.

August 21 (fifth day). Pulse 112. Very restless and delirious in the night; but intelligence is now clear. Three lenticular rose-spots on chest. Tongue dry, red, and glazed. Abdomen distended and tympanitic, but not tender. No stool since admission. Was ordered milk and beef-tea, and one teaspoonful of castor-oil.

August 22 (sixth day). Last night, the patient again became very restless and delirious, and to-day he is quite unconscious, and very prostrate, but he has had no motion of the bowels. Wine and carbonate of ammonia were administered freely; but the patient gradually sank, and died at 1.30 p.m.

Autopsy, 49 hours after death.

Left side of heart firmly contracted; right side full of dark fluid blood; lungs healthy. Peyer's patches, and the solitary glands of the ileum were much elevated and indurated from morbid deposit. The surface of the patches was rough and granular, but no ulceration could be discovered. The edges of the patches, and the corresponding portions of peritoneum, were red and vascular, but there was no increased vascularity of the mucous membrane generally. The morbid changes in the intestinal glands were most developed towards the cæcum, and the mesenteric glands corresponding to this part, were much enlarged. The spleen was very large and soft. The kidneys appeared healthy; the bladder was greatly distended with urine.

CASE XXVIII.

Pythogenic Fever, with Acute Delirium and Pulmonary Complication.—Death about the 15th day.—Autopsy:—Ulcers in Ileum, with adherent Sloughs.—Enlargement of Spleen and Mesenteric Glands.—Lobular Pneumonia, etc.

William B——, aged 11, admitted into the London Fever Hospital, August 28, 1858. He had been ill for about twelve days, and in bed for a week. His chief complaints had been chilliness, loss of appetite, thirst, and headache. No diarrhœa, until day before admission, when he passed two watery stools. During the two nights preceding admission, he had been very delirious.

August 28 (thirteenth day). Pulse 112. Respirations 40; crepitation at bases of both lungs, and slight dulness at left base. Constant noisy delirium, and is with difficulty kept in bed. Pupils dilated. Skin very hot and dry. Temperature in axilla 104° Fahr. A circumscribed deep-pink flush on both cheeks. A good many lenticular spots on abdomen and back. Has been picking lips till they bleed. Tongue deep-red, dry, glazed, and fissured. Intense thirst. Abdomen distended and tympanitic, with gurgling and tenderness in right iliac fossa. No motion to-day. Was ordered beef-tea, milk, four ounces of wine, and turpentine-stupes to chest.

August 29 (fourteenth day). Pulse 112. Respirations 36. Is more prostrate. Urine passed in bed. Slept for a few hours last night, after two draughts containing ℥. x. Liq. Morph. Acet. Is still delirious, but is inclined to sleep. Pupils small. Several fresh spots. Tongue dry and brown along the centre; two watery motions. Was ordered six ounces of brandy.

The patient again became very noisy and delirious in the night. On the following morning, he was quite unconscious, and he died at 9.0 a.m.

Autopsy, 33 hours after death.

Rigidity well marked. Body moderately thin. No spots visible on skin. Veins on surface of brain empty over anterior half; full, posteriorly. No opacity of membranes. No sub-arachnoid fluid on hemispheres. Half-a-drachm of serum in each lateral ventricle, and about four drachms at base. Brain-substance firm; bloody points rather numerous.

Heart healthy. Slight hypostatic congestion of both lungs. Scattered through both lungs were a number of isolated nodules, about the size of hazel-nuts, the tissue of which was very hyperæmic, friable, and scarcely crepitant, but not granular on section. Right lung 12 ounces; left 13 ounces. Peritoneum contained about six ounces of clear serum. Stomach healthy. The six Peyer's patches nearest to the cæcum were indurated, and raised one-tenth of an inch above the mucous membrane. The peritoneum corresponding to these patches was brightly injected. The solitary glands in the lower four inches of the ileum, and in the cæcum and ascending colon, were also much enlarged from morbid deposit. Ulceration had commenced in all of the diseased Peyer's patches, and in most of the solitary glands, but the surfaces of all the ulcers were covered with yellowish-brown sloughs, still firmly-adherent. The small intestine was contracted and empty, and was invaginated to the length of about two inches, at three different places. The invaginations were readily reduced.

The colon was distended with gas. The mesenteric glands near the cæcum were much enlarged, some of them equalling a pigeon's egg in size. The spleen weighed 13 ounces. The gall-bladder contained pale watery bile. The kidneys were hyperæmic, but appeared healthy.

Case XXIX.

Pythogenic Fever with Acute Delirium and Pulmonary Complication. Death on 21st day. Autopsy: Lobular Pneumonia. Intestinal Ulceration very limited. Sloughs mostly detached. Enlargement of Spleen and Mesenteric Glands.

Harry B—, aged 19, was admitted into London Fever Hospital, October 6th, 1858. Not much information could be obtained respecting his state before admission, except that he had been ill for about fifteen days, and in bed for a week; that his bowels had been relaxed, and that he had been delirious for several days.

October 7th (17th day). Pulse 108. Was very noisy and delirious in the night; but to-day answers when spoken to; pupils much dilated. Skin hot and dry; several lenticular rose-spots on abdomen. Tongue moist and red at tip. Abdomen tender and tympanitic; two light, watery stools. Was ordered mistura camphoræ ,beef-tea, milk, and four ounces of wine.

October 8th. Pulse 96. Very delirious in the night. Tongue dry, red, glazed, and fissured; three stools.

October 10th (20th day). This morning became rather suddenly worse. Pulse 120. Respirations 36; moist râles all over chest, but no decided dulness. Was very noisy and delirious in the night, but to-day is rather drowsy, with occasional muttering delirium. Pupils less dilated, but still larger than natural. There is a circumscribed flush on both cheeks of an almost livid hue. Several fresh spots. Tongue clean, red, glazed, and fissured. Much tenderness in right iliac region; one stool. Was ordered eight ounces of brandy, and a mixture containing ammonia and chloric ether.

The patient died at 5 a.m. on the following morning. Was quite unconscious for several hours before death.

Autopsy 29 hours after death.

Limbs rigid. No spots visible on skin. Right lung 30 ounces; left 24 ounces. Both lungs, but especially right, contained a number of circumscribed nodules of granular consolidation, varying in size from a pea to a walnut.

The greater portion of the small intestine, and the whole of the colon were healthy. The disease was limited to the lower eight inches of the ileum. The mucous membrane, for three or four inches above the valve, was one mass of ulceration, the surface of which was clean, and the edges not at all thickened. Above this, were several small ulcers, none larger than a sixpence, the surfaces of which were likewise clean, and in which cicatrization appeared to have commenced. To one only of these ulcers a yellowish slough was loosely adherent. Mesenteric glands much con-

gested and enlarged, but none of them exceeded a cherry in size. Spleen 17 ounces, dark and soft. Gall-bladder distended with a thin, almost colourless, fluid. Kidneys enlarged, each weighing 6 ounces; capsules non-adherent; surfaces smooth; cortical substance hypertrophied.

Case XXX.

Pythogenic Fever, at first of a mild character and simulating Remittent Fever. Pulse remarkably quick. Acute Delirium about 26th day, followed by typhoid symptoms, and death about 30th day. Autopsy: Ulcers of Ileum in process of reparation. Mesenteric Glands and Spleen only slightly enlarged.

Margaret C—, aged 21, was admitted into Fever Hospital, September 22nd, 1858. Cannot state the precise day on which she began to feel ill; but it was about eleven days before admission. Has only kept her bed for one day. Her chief symptoms have been weakness, loss of appetite, pains in the limbs, and feverishness towards night.

September 23rd. Pulse 108 in the morning, and 120 in the afternoon. Was restless in the night and slept badly, but intelligence is clear and expression almost natural. Skin hot. No eruption. Tongue moist and furred, very red at the edges; abdomen distended and tympanitic, with considerable tenderness. Five light watery stools. Was ordered milk and beef-tea, and a mixture containing Acetate of Lead (gr. iij.) and Liq. Morph. Acet. (mxiij.) after each motion of the bowels.

September 25th. Pulse 120 in the afternoon, but in the morning it was only 90. Intelligence clear; slept at intervals; no delirium; eyelids drooping and pupils dilated; expression languid. Temperature under tongue 104° Fahr.; a circumscribed flush on left cheek; two indistinct lenticular spots on back. Tongue moist and almost clean, red at the tip; occasional vomiting of bilious fluid; one liquid stool. Urine 19 ounces, rather dark, and contains 422 grains of urea. Was ordered four ounces of wine, a mixture containing carbonate of ammonia, and turpentine-stupes to the abdomen.

For the following week, the patient continued much in the same state. The pulse varied much, but was always quickest in the afternoon, when it was sometimes as high as 130. The intelligence remained clear, and the patient slept tolerably well, but the pupils were dilated and the eyelids drooped. No more spots could be discovered, although they were looked for daily; but there was usually a circumscribed pink flush on one or both cheeks. The skin was usually moist in the morning, and dry in the afternoon; in the night, there was usually considerable perspiration. There was occasional vomiting and slight diarrhœa.

October 2nd (22nd day). Is not so well. Pulse 150 in afternoon. Did not sleep so well, but intelligence is clear; pupils large. Temperature 105¼° under tongue. Has been perspiring very much, and to-day there are several distinct lenticular rose-spots on back. Tongue moist and furred, red at the edges; two light watery stools. Was ordered six ounces of brandy.

October 3rd. Pulse 154. Had slight delirium in the night, and is rather drowsy, but intelligence is clear. Insists that she is going to die. Pupils natural; skin dry, but perspired in the night; three or four fresh spots on abdomen. Tongue dry and red along the centre; no motion of bowels. Temperature 104½°; urine 25½ ounces, containing 352 grains of urea.

October 5th. Pulse 160; respirations 28; slept after opiate draught and has had no return of delirium. Several fresh spots; tongue dry and red; no motion for three days; urine 21¾ ounces, containing 490 grains of urea; brandy increased to 8 ounces, and was ordered a mixture containing ammonia and chloric ether.

October 7th (27th day). Pulse upwards of 160; respirations 36; is very prostrate. Was very restless in the night, and occasionally talked nonsense. To-day is dull and stupid; pupils rather small; is sweating profusely, and has copious sudamina on chest and abdomen; several fresh spots; tongue dry, red, and clean; occasional sickness, and brings up a yellowish liquid; bowels opened by an enema; urine 17 ounces, containing 394 grains of urea. Brandy increased to 12 ounces.

In the night of October 7th, the patient became violently delirious, and on October 8th, she was scarcely conscious. Constant floccitatio and occasional subsultus, urine passed in bed; pupils again dilated; eyes open and staring. Continued in the same state until the evening of October 11th, when the carphology ceased, and she became much more conscious, and bade adieu to her sister, telling her she would be dead before morning. Pulse too quick to count; respirations 46; a few fresh spots.

October 12th. Gradually sank, and died at 8½ a.m.

Autopsy 29 hours after death.

Slight rigidity; no spots visible on skin; body much emaciated.

Arachnoid slightly raised above convolutions by a considerable amount of serosity. Two drachms of serous fluid in each lateral ventricle, and more than half an ounce at base. No abnormal vascularity of pia mater or of brain-substance. Brain 50 ounces, firm.

Right lung 15 ounces, and left 15½ ounces. Indications of old tubercle, at both apices; but lungs, in other respects, healthy. Heart 7 ounces. Right cavities filled with a dark clot, like currant-jelly; left empty.

Stomach healthy. Numerous ulcers in lower three feet of ileum, corresponding to Peyer's patches. The largest were near the cæcum. There was no induration or thickening at the bases or edges of these ulcers. Some of them were surrounded by a loose fringe of mucous membrane, but in others the mucous membrane appeared continuous with the surface of the ulcer. One ulcer, more than two feet above the valve, had its edge considerably thickened, and a large yellowish-brown slough loosely-adherent to its surface, and two other ulcers in this situation had minute specks of slough still adhering to them. Many of the solitary glands were likewise ulcerated. The mesenteric glands were enlarged, but none

exceeded a hazel-nut in size. The spleen weighed only 5½ ounces. The kidneys were healthy; the gall-bladder was distended with a limpid, almost colourless liquid, containing white flakes.

c.—Analysis of Principal Symptoms.

a. The Physiognomy.

The heavy, stupid expression, so characteristic of typhus, is comparatively rare in pythogenic fever. Many patients pass through this disease without any remarkable change of countenance. Even when they are unable to give coherent answers, the countenance may be little altered, with the exception of dilatation of the pupils, and an expression of languor, ennui, or sadness. But in severe cases, when the disease assumes the 'typhoid state,' the *facies typhosa* may be indistinguishable from that of typhus (see page 127). The countenance rarely presents that general flushing of a dusky tint, so common in typhus; but it is either abnormally pale, or there is a circumscribed pink flush on one or both cheeks, which I have never seen in typhus. This flush was present in 74 of 100 cases, of which I have careful notes. Jenner met with it in 11 of 23 fatal cases.[i] It varies in intensity in different patients, and in the same patient at different times—it often disappears entirely, and returns; as a rule, it is most marked in the afternoon and evening, and after the administration of food or stimulants. It occurs in mild as well as severe cases; but, in the latter, it is usually of a deeper tint than in the former. Associated, as this appearance often is in the advanced stages of the disease, with great emaciation, sunken eyes, and rapid breathing, it forcibly recalls the aspect of persons in the hectic stage of pulmonary phthisis.

b. Symptoms referrible to the Skin.

1. *The Eruption* of pythogenic fever consists of isolated lenticular spots—the '*tâches roses lenticulaires*' of Louis (see Plates III IV. and V). Their colour is rose or pink, but varies slightly in tint, according to that of the patient's skin. Their form is rounded and regular; their margin is well-defined; and they measure from half a line to two lines in diameter. When the point of the finger is passed gently along the skin, each spot can in most cases be felt slightly elevated above the surface. Their outline is rounded and convex, but not acuminated. They are never indurated; but,

[i] Jenner, 1849 (2).

Plate II

Wertmüller del.

Lenticular spots of Pythagenic [illegible]

in very rare cases, a minute vesicle may be discovered at their apex.[j] They are never converted into petechiæ; but during the whole period of their existence they disappear completely on pressure, and return when the pressure is removed. They are never observed on the dead body. They are developed in successive crops, each spot lasting three, four, or five days, and then fading, while fresh spots continue to appear. I have verified this observation hundreds of times, by surrounding daily every fresh spot with a circle of ink, and writing the date of each on the skin.

The number of these spots is usually small; and hence they are often over-looked. In most cases, the number present at one time does not exceed twenty or thirty; and sometimes three or four spots are all that can be discovered. Occasionally, however, the spots are very numerous, and then the edges of two contiguous spots may cohere. Still this is a rare circumstance, and the spots are never seen to merge into irregular patches, as occurs in typhus. Of 98 cases in which I have noticed the eruption, the number of spots present at one time never exceeded 20 in 61; in 37 cases it exceeded 20; and in 9 cases it exceeded 100. In 1 of the cases I counted upwards of 1000 spots at one time (see Case XXXI and Plate IV); and two other cases where they were equally numerous, have come under my notice. According to Barthez and Rilliet, with whose experience my own concurs, the spots are less numerous in young children than in adults. In most cases, these observers did not notice more than 6 at one time, and rarely so many as 20; in only 1 of 111 cases did they find them very numerous.[k]

The most common situations of the spots are on the front of the chest and abdomen, and on the back. I have often succeeded in finding them on the back, when they existed nowhere else; and I have occasionally noted that they were larger and more numerous on the back than in front. This circumstance may be due to the greater warmth of the skin of the back, on which the patient lies. Louis and Jenner mention instances where the spots were developed in large numbers after a warm bath, and I have known them to appear first on a portion of skin to which a sinapism had been applied. In 8 of 98 cases, I have noted the spots as present on the arms or legs, and in 1 case on the face.

The date of appearance of the spots is between the seventh and

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[j] I have only noticed this appearance in two or three cases. Jenner, Peacock, and W. T. Gairdner have also observed a minute vesicle, at the apex of the spots, in exceptional cases See JENNER, 1853, p. 285; PEACOCK, 1856 (No. 1); GAIRDNER, 1862 (No. 2), p. 125.

[k] BARTHEZ and RILLIET, 1853, ii. p. 684.

twelfth days (inclusive). It was so in 39 of 46 cases, in which I noted the point; in 4 cases only were the spots present before the sixth day, and then they appeared on the fifth day. On the other hand, in 3 cases they were not seen before the fourteenth day; and in 1 case, they did not appear until the twentieth day. In none of Chomel's cases was the eruption observed before the sixth day;[l] and only in 1 case did Louis meet with it, as early as the fifth day.[m] In children, it appears rather earlier than in adults: Taupin, Barthez and Rilliet state that it occurs occasionally as early as the fourth day.[n]

The duration of the eruption varies from seven to twenty-one days, according to the date of its first appearance and the total duration of the fever. As a rule, it disappears with the commencement of convalescence; but in certain cases, the spots continue to come out after the general symptoms have begun to improve, and the patient is apparently convalescent. It is well to be aware of this circumstance, for as long as the spots continue, a slight indiscretion may bring on an exacerbation of the febrile symptoms. The mean duration of the eruption, in 30 cases which recovered under my care, and in which it was watched, either from its first appearance, or from the eighth day of the disease, was $14\frac{1}{2}$ days, the shortest period being 4 days, and the longest 35. In children, according to Barthez and Rilliet, the eruption rarely lasts longer than 7 or 8 days (see also page 500).

The eruption is not invariably present, but is perhaps more common than is generally believed.[o] Of 1820 cases admitted into the London Fever Hospital during ten years, it was noted in all except 224, or 12·3 per cent.; and in many of the 224 cases, the fact of the spots not being observed was probably due to their not having been looked for with sufficient care. Of 142 cases, of which I have notes, the spots were discovered in all but 7. Louis observed them in 160 out of 177 cases; and in the remaining 17 cases, with the exception of 5, he was unable to state positively that they were wholly absent.[p] In America, Bartlett rarely failed to find them, when they were properly sought for.[q] They are more frequently absent in patients over 30 and under 10 years of age, than in patients between 10 and 30. Of 1413 cases admitted into the Fever Hospital, between the ages of 10 and 30, the eruption was observed in all except 142, or 10 per

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[l] Chomel, 1834. [m] Louis, 1841, ii. 105.
[n] Taupin, 1839; Barthez and Rilliet, 1853, ii. 683.
[o] Peacock failed to find them in 9 out of 62 cases. (Peacock, 1856, No. 1). According to Parkes, they are absent in 20 per cent. (*Assoc. Med. Journ.* 1856, p. 993.) [p] Louis, 1841, ii. pp. 96-105, [q] Bartlett, 1856, p. 63.

cent.; of 252 patients over 30, it was not observed in 40, or in nearly 16 per cent.; and of 107 cases under 10, it was not noted in 37, or 34½ per cent. Jenner believed that the spots were comparatively rare above 30 years of age. Barthez and Rilliet failed to find them in one-fourth of 111 cases in young children; Taupin, on the other hand, noted them in 110 out of 121 children, and believed that they were as common in early, as in adult life. As to sex, the eruption was unobserved in 127 of 905 males admitted into the London Fever Hospital; but in only 97 of 915 females.

There is no relation, as in typhus, between the presence or abundance of the eruption, and the severity of the disease. Of 37 cases in which I noted the spots as exceeding twenty, 7 died; and of 61 cases where the spots never amounted to twenty, 12 died; so that the rate of mortality, in the two classes, was almost equal. Some writers, indeed, have regarded a copious eruption as a favourable sign, rather than otherwise.[r] Louis found the eruption more scanty, and oftener absent, in fatal cases than in those which recovered. Barthez and Rilliet are of opinion, that in children the spots are fewer and oftener absent in the severe cases than in the mild. Dietl, of Cracow, who has paid particular attention to the subject, thinks that the prognosis is usually most favourable in those cases where the eruption is most abundant.[s]

The following are the principal points of distinction between the spots of Pythogenic Fever and those of Typhus.

Pythogenic Fever.	*Typhus.*
1. Pink or rose-coloured throughout.	1. May be dirty-pink or red at first, but soon become reddish-brown.
2. Undergo no change, until they fade or disappear. Never converted into petechiæ.	2. Become gradually darker, and are often converted into petechiæ.
3. Circular.	3. Of irregular form.
4. Isolated and few in number.	4. Numerous and adhere in patches.
5. No subcutaneous mottling.	5. Mottling common, in addition to spots.
6. Elevated above the skin.	6. Not elevated, except at first appearance.
7. Disappear on pressure, as long as they last.	7. Do not disappear on pressure, except at first.
8. Appear on 4th or 5th day.	8. Rarely before 7th day.
9. Appear in successive crops.	9. Never in successive crops.
10. Each spot lasts only three or four days.	10. Many of the spots may last to the end of the fever.
11. Never present on dead body.	11. Often persist after death.
12. A large number does not indicate danger.	12. Direct ratio between the number and darkness of the spots and the severity of the case.

It is important to determine whether the lenticular spots above

[r] See STEWART, 1840, p. 326.

[s] *Edin. Med. Journ.* 1856, ii. 365.

described, be ever present in other diseases, besides Pythogenic Fever. Louis, in the first edition of his work (1829), stated that he had found them in 12 cases of other acute diseases; but in the second edition (1841), he observed, that, in the intervening twelve years, he had sought for them in vain in every disease but 'typhoid 'fever,' and he was inclined to think that he had formerly mistaken ordinary pimples for the lenticular spots.[t] Possibly, other observers have committed a similar error. Dr. Waller, of Prague, and Messrs. Barthez and Rilliet, state that they have met with lenticular rose-spots in cases of acute phthisis:[u] but their observations require confirmation. I have always failed to find them in such cases. From my own observation, I feel convinced that an eruption, which presents all the characters above mentioned, is peculiar to Pythogenic Fever.

Case XXXI.

Pythogenic Fever, with very Copious Eruption.

Mary E——, aged 20, servant in a private family, was admitted into the London Fever Hospital, September 16, 1858. Her illness had commenced about ten days before admission, with giddiness, nausea and vomiting, pain in the abdomen, and loss of appetite. From the first, the bowels had been open two or three times a-day.

September 17 (twelfth day). Pulse 120. No headache. Intelligence good, but sleep very disturbed; physiognomy languid, but not at all stupid; pupils large. Skin hot; a circumscribed pink flush on one cheek. Two or three lenticular rose-spots on abdomen. Tongue moist and furred, and red at edges; four ochrey watery stools. Was ordered beef-tea and milk.

September 18 (thirteenth day). Pulse 114; weak. Temperature under tongue 104½° Fahr. Lenticular spots very numerous on chest, abdomen, and arms; more than 200 were counted on chest and abdomen alone. Belly tympanitic; gurgling and tenderness in right iliac fossa; four stools. Was ordered four ounces of wine, and acetate of lead (gr. iij.) after each motion.

September 19 (fourteenth day). Pulse 116. Is quieter and a little drowsy; but intelligence clear, and has no delirium. Pupils dilated, and is rather deaf. Sixty fresh spots have appeared on front of chest and abdomen, and they are also very numerous on back, arms, and legs, and there are even a few on hands, feet, and face.

September 21 (sixteenth day). Pulse 124. Is more prostrate, and has slight tremor of the hands. Intelligence clear; but had slight delirium in the night. Circumscribed deep-pink flush on both cheeks. Lenticular spots still very numerous. During the last two days, 160 fresh ones have

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[t] Louis, 1841, ii. p. 107.

[u] See Jenner, 1853, p. 465; also Barthez and Rilliet, 1853, ii. 684, 697.

appeared on the chest and abdomen alone, while several of those marked on September 18, are no longer visible. A few of the spots are fully a fifth of an inch in diameter; they are all elevated and rounded, and disappear completely on pressure. Although mostly isolated, two spots might be seen, here and there, with their edges in contact. The spots were calculated to exceed one thousand; their appearance on the abdomen is represented in Plate IV. Lips dry and cracked; tongue red and moist; six stools. Was ordered eight ounces of wine, and chalk mixture with catechu.

September 23 (eighteenth day). Pulse 120. More drowsy, and has occasional delirium; pupils rather large. Temperature under tongue 103¼° Fahr. Ninety fresh spots on chest and abdomen since September 21, and many of those previously marked have disappeared; still several on face. Four light, watery stools. Was ordered a mixture every four hours, containing acetate of lead (gr. iij.) and liq. morph. acet. (ɱ iij), also a starch and opium enema at night.

September 25 (twentieth day). Pulse 112. Answers when spoken to, but is very drowsy and confused; pupils rather small. Temperature under tongue 102½° Fahr. Fifty fresh spots on chest and abdomen. Two stools. More prostrate, and tremors increased. Ordered eight ounces of brandy.

September 26. Twelve fresh spots on chest and abdomen. Tongue red and dry. No stool since yesterday morning.

September 28 (twenty-third day). Pulse 114. Is more conscious. Temperature under tongue 101¾° Fahr. Spots much less numerous, and only twelve fresh ones on chest and abdomen in last two days. One stool.

September 30 (twenty-fifth day). Pulse 96. All the symptoms have improved. Temperature under tongue 99½° Fahr. Spots less numerous, and only three fresh ones on chest and abdomen. Tongue moist and smooth; no stool. Yesterday passed 70 fluid-ounces of urine, containing 496 grains of urea, and to-day 43¾ fluid-ounces, containing 575 grains. Wine was substituted for brandy.

October 2. Pulse rose to 108 yesterday, but is to-day 84. Feels and looks much better. No fresh spots, and only six or seven of the old spots remain on front of chest and abdomen. Temperature 99½° Fahr.

October 5 (thirtieth day). Pulse 80. Temperature 98° Fahr. Tongue clean and moist, One formed stool daily. Only a few traces of spots on back. Convalescent.

October 22. Discharged from the Hospital, well.

2. *Scarlet Rash.* Now and then the appearance of the lenticular spots is preceded, for two or three days, by a delicate scarlet rash, all over the body (see Case XXV.) I have noted this rash in 5 out of 45 patients admitted into hospital within the first eight days of the disease, and in a sixth case, it was observed to precede the second eruption of spots, when the fever relapsed. Dr. Jenner

mentions an instance, where this rash co-existed with slight sore throat, and the disease was mistaken for scarlatina.[x] In the advanced stages of the fever, a red or purplish blush of the skin, disappearing on pressure, is occasionally observed over the dependent parts of the body.

3. *Petechiæ and Vibices* are met with in very rare cases. I only remember to have seen petechiæ twice. Trousseau records a case in which there were extensive vibices.[y] When petechiæ occur, they are not developed in the centre of the lenticular spots, but are independent.

4. *Tâches bleuâtres.* Spots of a delicate blue tint—the 'tâches bleuâtres' of French writers—are occasionally observed on the skin in cases of pythogenic fever. They are of an irregularly rounded form, and from three to eight lines in diameter. They are not in the least elevated above the skin, nor affected by pressure, even at their first appearance. They have a uniform tint throughout their extent, and they never pass through the successive stages, observed in the spots of typhus. Two or three of them are sometimes confluent. They are most common on the abdomen, back, and thighs, and in one instance I have seen them distributed along the course of the small subcutaneous veins (see Plate V.) The cases where I have met with these spots have usually been of a mild character; and Trousseau makes a similar observation.[z] They occur in other diseases, besides pythogenic fever.

5. *Sudamina* are alluded to by most writers. Louis observed them in 104 out of 141 adults; and Taupin in 104 of 121 children. They appear to be less common in England. Peacock met with them in only 22 of 52 cases; and Jenner in only 7 of 23 fatal cases. I have noted them in only 12 of 53 cases. Their most common situation is on the front of the chest or abdomen, or on the sides of the neck. They usually appear in the third or fourth week of the disease, along with perspiration. From the frequency with which Louis found sudamina, he was inclined to regard them as a specific character in 'typhoid fever,'[a] but they are probably equally common in all febrile diseases, attended with perspiration.

6. *Herpes* is occasionally observed on the lips.

7. *Desquamation* of the cuticle, on the cessation of the fever, is chiefly observed in cases where sudamina have been present; but

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[x] Jenner, 1850, xxii. p. 277. [y] Trousseau, 1861, p. 152.
[z] See Trousseau, 1861, p. 159; Forget, 1841, p. 226; Jenner, 1850, xxiii. 313.
[a] Louis, 1841, ii. 110.

in many other cases, the skin, during convalescence, is rough, from the separation of the cuticle in minute branny scales.

8. *The Temperature* is always more or less elevated. I have made observations with a delicate thermometer, held beneath the tongue, or in the axilla, in 26 cases. In only 1 case, did the temperature never reach 100° Fahr.; in 13, it reached 104°, or upwards; in 6, it was 105°, or upwards; and in 1, it was 108°. Many of the patients, moreover, did not come under observation, until after the tenth day of the disease. In most cases, I have found that the temperature is highest, during the first two weeks, and that after this, it diminishes slightly until convalescence, when it falls to the normal standard. It also varies slightly from day to day; but this circumstance is partly due to the observations being made at different hours. As a rule, the temperature rises one or two degrees towards evening, and falls in the morning. Wunderlich has published some interesting observations, showing that the temperature in abdominal typhus is always greatest in the evening, during the first two weeks, but that, in severe cases, it ultimately becomes more equable.[b] Thierfelder, also, has noticed a remission of the temperature in the morning.[c] Those cases, in which the temperature is highest in the early stage, are usually afterwards the most severe.

9. *Moisture.* The skin is usually dry, as well as warm; but in most cases, which have come under my notice, the dryness has alternated throughout the fever with clamminess, or actual perspiration; and in 19 of 84 cases, I have noted the perspiration, as considerable in quantity. Perspiration usually occurs during the night, the skin, in the day-time, being dry.

10. *Odour.* There is rarely any peculiar odour given off by the skin in pythogenic fever (see page 134). An experienced nurse at the Fever Hospital once informed me, that she could always distinguish the typhus from the enteric cases, by the peculiar odour given off by the former, which was absent in the latter.

11. *Bed-Sores* (see *Complications.*)

c. Symptoms referrible to the Circulating System.

1. *The Pulse* is accelerated. Out of 100 cases, I ascertained that it exceeded the normal standard, at some time of the fever, in all but one; in 97 cases, it exceeded 90; in 85 cases, it exceeded 100; in 70 cases, it exceeded 110; in 32 cases, it exceeded 120; in 25 cases, it exceeded 130; in 10 cases, it was above 140; and in 2, above 150.

[b] WUNDERLICH, 1858, p. 19. [c] THIERFELDER, 1855.

Its frequency, however, varies at different times, in the same case. Thus, in four cases, the pulse, on several successive days of the fever, was noted as follows :—

Day of Fever.	Fem. æt. 23.	Fem. æt. 30.	Fem. æt. 18.	Male æt. 21.
10	...	...	96	...
11	...	...	120	...
12	132	120	88	108
13	96	96	100	92
14	116	120	80	96
15	120	100	72	96
16	124	96	60	120
17	120	104	72	88
18	132	96	84	90
19	96	108	80	120
20	120	120	120	108

These variations in the pulse of pythogenic fever, are also referred to by Jenner;[d] they are independent of any change of treatment —a circumstance to be borne in mind, in drawing conclusions from the pulse as to the effects of remedies.

In mild cases, and in the earlier stages of more severe forms of the disease, I have often found the pulse ten or more beats higher at one period of the day than at another; as a rule, it is lowest in the morning. Similar observations have been recorded by Traube, Parkes, and H. Weber.[e] These changes of the pulse, as well as of the temperature and of the degree of moisture of the skin, often impart a more or less remittent character to pythogenic fever. Some cases, indeed, run a course extremely similar to that of remittent fever. The variations in question, however, become less marked in the advanced stages of severe cases.

With the commencement of convalescence, the pulse falls slowly; there is rarely any sudden reduction. Its frequency, however, may be kept up, while amendment goes on, owing to complications, or to mere weakness and nervous irritability. From these causes, it may be even more rapid in convalescence, than during the fever.

At the same time, it is remarkable how low the pulse sometimes falls, even while the fever continues, as shown by the eruption of fresh spots. In 6 out of 100 cases, I found it fall to 60; in 2 other cases, to 56; and in a 9th case, to 52. In another case, it fell to 37, and never, throughout the fever, exceeded 56, but it rose with convalescence to 66. My impression is, that an abnormally slow pulse during convalescence is much rarer than in typhus.

[d] JENNER, 1853, p. 235. [e] See TRAUBE, 1853, *Rev.* pp. 42-3.

As a rule, those cases are most severe, in which the pulse is quickest. Thus, of 30 cases, where I found the pulse never exceeded 110, not one died; whereas, of 70 cases, where it was above 110, 21, or 30 per cent., died; of 32 cases, where it was above 120, 15, or 47 per cent., died; of 25 cases, where it was above 130, 13, or 52 per cent., died; and of 10 cases where it was above 140, 6 died. Two of the patients who recovered, after the pulse had exceeded 140, were under ten years of age. Still, I have known cases prove fatal, where the pulse never reached 100; and in 8 of Louis's fatal cases, the pulse never exceeded 90.[f]

During the first week or ten days of the disease, the pulse often exhibits considerable resistance; but after this, or sometimes earlier, it is soft and compressible; and, in the advanced stage, it may be small, feeble, irregular, intermittent, or imperceptible. Louis noted the pulse as irregular or intermittent in 7 out of 41 fatal cases, and in 6 out of 57 severe cases, which recovered.

2. *Action of the Heart.* Impairment, or complete absence, of the impulse and first sound of the heart are observed in some severe cases, but are rarer phenomena than in typhus. For further details, the reader is referred to page 136.

3. *Blood* (see *Post-Mortem Appearances*).

d. Morbid Phenomena of the Respiratory System.

1. *The respiratory movements,* in the advanced stages, are usually quickened, independently of pulmonary complications. Of 60 cases, in which they were counted daily, I found that they exceeded 20 in the minute, in 50; 30, in 38; and 40, in 22; but in most of the cases, where they exceeded 40, and in some others also, the lungs were diseased. The respirations vary with the pulse, but in those cases where the pulse is remarkably slow, there may be no corresponding diminution in the rate of respiration. Thus, in one case, the pulse was 64, and the respirations 28; in another, the pulse was 58, and the respirations 26; and in a third case, the pulse was 42, and the respirations 48, although no pulmonary lesion could be discovered. Occasionally, the breathing is irregular, noisy, or 'nervous,' as described under the head of typhus (page 137).

2. *The expired air* has not yet been carefully examined; but in severe cases, during the typhoid stage, the breath is very offensive, and probably contains an increased amount of ammonia, as in typhus.

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[f] Louis, 1841, ii. 347.

e. Morbid Phenomena presented by the Digestive Organs.

1. *The Tongue*, at first, is moist, and covered with a thin white fur, while its tip and margin are unusually red. It may remain in this state throughout the attack; or, about the end of the second week, it may become dry and brownish along the centre, and afterwards it may be covered with a thick, brownish crust, or it may become clean, red, dry, glazed, and fissured.

Cases may prove fatal where the tongue is never brown. Of 45 in 100 cases, where I noted the tongue as dry and brown, 16 died; but of the remainder, 5 were fatal, in which the tongue was never dry and brown. The tongue also was moist throughout, in 16 of 40 fatal cases recorded by Louis, and in 6 of 20 fatal cases noted by Jenner.[g]

A peculiarity of the tongue is its unusual redness, which may be confined to the tip and edges, or extend over the entire surface. I have noted unusual redness in 69 out of 100 cases. In 16 of the 69 cases, 5 of which were fatal, the entire tongue was red, and its surface clean, smooth, and glazed. Jenner noted this glazed appearance in 5 out of 20 fatal cases. Occasionally, I have seen the tongue of a bright scarlet hue, with enlarged papillæ, as occurs in scarlatina.

Another character of the tongue is the existence of transverse fissures, often deep, and causing much pain. They were noted in 35 of my 100 cases, and in 4 of 20 fatal cases observed by Jenner. Louis mentions cases, where they proceeded to extensive ulceration, with great thickening of the tongue.

Inability to protrude the tongue is much rarer, even in fatal cases, than in typhus.

2. *Lips and Teeth.* The lips are usually parched, and, in severe cases, may crack and bleed, a condition which, in children, is often aggravated by picking. When the typhoid stage is developed, sordes collect on the teeth. In rare cases, hæmorrhage from the gums occurs.

3. *The Appetite* is usually lost, but, in mild cases, it may remain tolerably good throughout the disease. I have noted it, as remaining in 11 out of 100 cases.

4. *Thirst* is an almost invariable symptom in the early stages. In 39 out of 100 cases, I have noted it as excessive.

5. *Dysphagia* (see *Pharyngitis*, under *Complications*).

6. *Nausea* and *sickness* are very common symptoms. Nausea

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[g] Louis, 1841, i. 474; Jenner, 1849 (2).

exists in many cases; and, in 36 out of 100 cases, I have noted vomiting. In 12 out of 63 of the cases, it was one of the earliest symptoms;[h] in the others, it came on after the first week. In most of the cases, the vomiting was only occasional; but in 8, it was protracted and distressing. It was usually associated with some pain and tenderness at the epigastrium. Louis observed vomiting in 36 out of 108 cases, and epigastric pain or tenderness in 59 out of 110 cases.[i] Vomiting, at the commencement of the disease, I am inclined to regard as a rather favourable symptom. In several cases, where it was very urgent, I have known the disease afterwards run a mild course. But when vomiting comes on suddenly after the second week, it is sometimes the first symptom of peritonitis from perforation.

The vomited matter usually consists of a greenish bilious fluid. Chomel mentions one case, where it contained blood;[k] and in one instance, I have known fæcal vomiting persist for 36 hours before death, which was due to perforation of the bowel.

7. *Meteorism* is observed in most cases. Out of 100 cases, I found that the abdomen was unusually resonant or distended at some period of the fever in 79; and in 17, the distension was great. Louis noted meteorism in 89 out of 134 cases.[l] In one fatal case, he observed it as early as the third day; but, as a rule, it does not supervene until after the first week. It is most developed in grave cases. Thus, in 21 fatal cases, I noted it in 20; while Jenner observed it in 18 out of 19 fatal cases. Of 17 cases in which I have noticed extreme tympanitis, death occurred in 7, while of 62, in which it was moderate or slight, only 14 died; and of 21, when it was absent, none died. Louis noticed great meteorism in one half of his fatal cases; but only in 7 of 88 cases, which recovered. The distension is of a peculiar form, the convexity being from side to side, in place of from above downwards, owing to the flatus being chiefly contained in the colon. Unlike the meteorism of typhus (page 141), that of pythogenic fever is almost invariably associated with pain and tenderness of the abdomen, and diarrhœa.

8. *Gurgling* is perceived in most cases, when pressure is made rather abruptly in the right iliac region. According to Chomel, it is much more common in pythogenic fever than in ordinary diarrhœa. I have noted it in 31 out of 44 cases.

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[h] In 37 of the 100 cases, it was not noted whether there had been any vomiting before admission. [i] Louis, 1841, i. 459. [k] Chomel, 1834, Case x.
[l] Louis, 1841, i. 452,

9. *Abdominal Pain or Tenderness* is rarely absent. Patients often complain of pain in the abdomen; and only in exceptional cases, is tenderness not elicited, when pressure is made in the right iliac region. I found tenderness at this part, in 71 of 81 cases; 16 of the 71 died, but none of the remaining 10: of 5 patients, who complained of severe pain, at the commencement of the disease, 3 died. Louis noted abdominal pain, in 106 out of 127 cases; of 39 fatal cases, it was present in all, and in 16, on the first day; whereas, of 31 mild cases, it was absent in 10, and in only 4, existed on the first day.[m] Jenner noted abdominal pain in 15 out of 20 fatal cases.[n]

10. *The Spleen* is often much enlarged, especially in young persons. In children, Taupin discovered considerable enlargement of the spleen in 109 out of 121 cases;[o] but Barthez and Rilliet only met with it in 28 out of 105 cases.[p]

11. *Diarrhœa* is the rule in pythogenic fever; and constipation the exception. I have noted diarrhœa in 93 out of 100 cases, and M. Barth observed it 96 out of 101 cases at Paris.[q]

Its intensity varies. In 23 of 84 cases, I found that the motions never exceeded three in the day; in 51, they numbered four or upwards; and in 19, they exceeded six. In several cases, I have known them amount to twelve, or more. There is no relation between the intensity of the diarrhœa and the extent of the intestinal mischief.

The date of commencement, and the duration of the diarrhœa vary. Diarrhœa may be one of the first symptoms, as happened in 38 of my 100 cases, and then it may cease after a few days, and not return. At other times, the bowels are at first confined, and urgent diarrhœa follows the administration of an ordinary purgative. In some cases, diarrhœa does not commence, until the third or fourth week of the disease, and then it may be profuse.

Louis ascertained that those cases were most severe and fatal, where the diarrhœa was most persistent and urgent; and this conclusion is borne out by my experience. Of 34 of my cases, where the diarrhœa, from its severity or duration, was noted as excessive, 10 died; but only 10 died out of 59 cases, in which the diarrhœa was moderate or slight.

Diarrhœa is not invariably present (see Cases XXV. and XXVII.) In 7 of my 100 cases, it existed at no stage of the disease; and in 4, there was constipation. Bartlett and Flint

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[m] Louis 1841, i. 445. [n] Jenner, 1849 (2).
[o] Taupin, 1839. [p] Barthez and Rilliet, 1853, ii. 677. [q] Louis, 1841, i. 439.

speak of diarrhœa as often absent in mild cases;[r] but one of the 7 cases above referred to was fatal. There was also no diarrhœa, in 3 fatal cases recorded by Louis, and in 2, observed by Jenner. Diarrhœa may be absent in cases where the ulceration is most extensive and advancing to perforation. Jenner records the case of a female, whose bowels had all along been confined, but who died on the twenty-fifth day of profuse hæmorrhage from the bowels.[s] Wilks mentions the case of a girl who died at the end of the third week; her bowels had been confined, and, after death, the small intestines were found filled with firm scybala, with an ulcer beneath each.[t] In several cases, where a relapse has occurred, I have known the bowels constipated throughout the primary attack, and much relaxed during the relapse.

12. *The Characters of the Stools,* in pythogenic fever, are peculiar. They are liquid, and of the colour of yellow-ochre; their odour is very offensive and often ammoniacal; and their reaction is alkaline, whereas the fæces in health are always acid. Simon, Marklein, Parkes,[u] and Lehmann[x], all attest their alkaline character, and I have repeatedly confirmed their observations; according to Parkes, it is due both to carbonate of ammonia, and a fixed alkali. On standing, the stool separates into two layers,—a supernatant fluid, and a flaky sediment. The former has a yellowish, or pale-brown colour; its specific gravity is about 1015, and it contains about 40 parts in 1000 of solid matter, which consists chiefly of albumen and soluble salts, particularly chloride of sodium.[y] The deposit is made up of particles of undigested food, disintegrating intestinal epithelium and blood-corpuscles, shreds of sloughs, which have separated from the intestinal ulcers, and multitudes of crystals of triple phosphate. The existence of crystals of triple phosphate in the stools of 'abdominal typhus,' was pointed out by Schönlein, in 1835,[z] and has been referred to by Gluge,[a] Parkes, and many other observers. Schönlein imagined that they were peculiar to this disease; but they are now known to be abundant in all diseases, where, as in pythogenic fever, the stools have a marked tendency to decomposition.

The characters of the stools, now described, are best seen after the tenth day of the disease. In some cases, the stools, in place of being watery, are pultaceous, frothy, as if fermenting, and so light as to float on water; at other times, I have known them to resemble mud or bird-lime; or they may contain blood.

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[r] Flint, 1852; Bartlett, 1856, p. 58. [s] Jenner, 1849 (2) [t] Wilks, 1855.
[u] Parkes, 1850, p. 396. [x] *Physiol. Chem.* Day's Transl. i. 150.
[y] Parkes, *loc. cit.* [z] Schönlein, 1836. [a] Gluge, 1837.

13. *Intestinal Hæmorrhage* is an important symptom. It occurred in 14 out of 84 of my cases; in 8 out of 134 cases noted by Louis,[b] and in 7 out of 21 fatal cases noted by Jenner.[c] In children, it appears to be rarer than in adults: out of 232 cases, under 15 years of age, observed by Messrs. Taupin, Rilliet and Barthez, it occurred only once.[d]

The quantity of blood lost may vary from a few drops to several pints. It is usually fluid; but sometimes, clotted. Its colour is sometimes dark, but often bright red, owing to the alkaline condition of the intestinal contents.

The source of the hæmorrhage varies. When it occurs after the twelfth day of the disease, and is copious, it is probably due to a small artery being laid open by one of the intestinal ulcers, or to a fungating condition of the morbid material in the Peyerian glands. In one case, Jenner found that water injected into the superior mesenteric artery, escaped freely from an ulcer in the ileum. At other times, the hæmorrhage appears to be due to rupture of the intestinal capillaries consequent on hyperæmia, or occasionally, perhaps, as suggested by Trousseau, to a liquefied condition of the blood; and then, the quantity may be slight. I have known intestinal hæmorrhage occur, on the fifth or sixth day of the disease, before ulceration had probably taken place; and Chomel mentions cases, where blood was found in the intestines, before ulceration had commenced.[e]

Different opinions have been expressed as to the importance of intestinal hæmorrhage, as regards prognosis. Bretonneau, Chomel, Louis, Jenner, Bell,[f] and most writers on fever have regarded it as a dangerous symptom. Six out of seven cases, where it occurred under the care of Chomel, proved fatal, and 3 out of 7 cases observed by Louis. On the other hand, Graves, in his Clinical Lectures,[g] speaks of certain cases where the occurrence of hæmorrhage was thought to be productive of marked benefit. More recently, Trousseau has taught that it is a less dangerous symptom than is generally thought, and has stated that during seven years, he had only known three cases, where it occurred, prove fatal.[h] The reporter of Trousseau's lecture adds, that in 115 cases of 'typhoid fever,' communicated to the French Academy by Ragaine, hæmorrhage occurred in 11, all of which recovered. Dr. H. Kennedy, of Dublin, also says that most cases recover, and that they are frequently benefited by the occurrence.[i] These statements have led some

[b] Louis, 1841, i. 433-9. [c] Jenner, 1849 (2).
[d] Barthez and Rilliet, 1853, ii. 705.
[e] Chomel, 1834. [f] Bell, 1860, viii. 390. [g] 1848, Vol. i. 266.
[h] Trousseau, 1859. p. 298; 1861, p. 149. [i] Kennedy, 1860, p. 226.

writers to regard intestinal hæmorrhage as a rather favourable symptom.[k] My experience is opposed to such a conclusion. When the hæmorrhage is scanty, it has probably little effect on the result; or before the twelfth day, it may do good by relieving intestinal congestion. But when profuse (and after the twelfth day, it is impossible to say that it will not be so), it is a most formidable symptom. Although I have known patients recover after profuse hæmorrhage, I have never observed the slightest benefit from it. On the contrary, I have repeatedly seen patients die unexpectedly by syncope a few hours after its occurrence, who had previously done well. Jenner,[l] and Dr. Joseph Bell make a similar remark. Five of my 14 cases proved fatal; and in 7 of the remainder the hæmorrhage was trifling. Graves himself says that excessive hæmorrhage must be carefully watched and checked, and Trousseau records one case, where the patient died, one hour after the commencement of the bleeding, which was so profuse, as to saturate the entire bedding.

f. Morbid Phenomena presented by the Urinary System.

1. *The Urine* has been carefully examined by many observers.[m]

The quantity, during the first week or ten days, is usually diminished. Sometimes it falls to one-half, to one fourth, or even to one-sixth of the normal standard, notwithstanding the increased amount of fluid ingesta. After the second week, the quantity increases, and I have frequently observed the urine to be copious, pale, and of low specific gravity, before the cessation of the fever. But in most cases, the quantity does not increase greatly until convalescence, when the urine is almost invariably very copious and of low specific gravity. At this period, I have repeatedly known as much as 80 or 90 fluid ounces passed in twenty-four hours.

The colour at first is darker than natural, and according to Vogel, it is more intense than can be accounted for by mere concentration.[n] Occasionally, in the advanced stages of the disease, and always in convalescence, I have found the colour unusually pale.

The acidity, during the first week or ten days, appears increased; but this is due to concentration of the urine; for, according to Parkes, when the exact amount of acid is determined by neutralizing

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[k] *Med. Times and Gaz.* 1859, ii. 361, 441. [l] Jenner, 1853, p. 286.

[m] For an able abstract of all that has been written on the subject, the reader is referred to Parkes, *On the Urine*, 1860, p. 244; see also Parkes, 1855.

[n] Neubauer's *Anleitung*, 2nd ed. p. 235.

with an alkali, it is found to be below the average, by one-fifth, or one-fourth. During the third and fourth weeks, the urine is very feebly acid, and is often alkaline, from decomposition of urea, or from the presence of fixed alkali.

The specific gravity varies. At first, when the urine is scanty, it may be from 1025 to 1030. Parkes, in one instance, found it as high as 1038. In the advanced stages, and particularly in the typhoid state, the specific gravity falls, and, in convalescence, I have often found it not to exceed 1005 or 1003.

The amount of urea excreted daily is always increased. The researches of J. Vogel,[o] A. Vogel,[p] Moos,[q] Brattler,[r] Warnecke,[s] Parkes,[t] Handfield Jones,[u] and others leave no doubt on the matter. Of 6 cases, where the urine was examined by myself and Dr. Sanderson, at the Fever Hospital, the urea was increased at one period of the fever, in all. The amount of increase varies. In most of Parkes's cases, the average increase was about one-fifth, *i. e.* the total quantity is about 480 grains, in place of 400. But occasionally, this amount is far exceeded. Alfred Vogel found as much as 78 grammes, or 1200 grains, passed in 24 hours; in one case, Parkes found 57 grammes, or 880 grains; and in one of my own cases, the quantity was 62·5 grammes, or 964 grains. Moos, Brattler and Warnecke, have shown that urea is excreted, in largest quantity, during the first week of the disease; and that after this, the quantity usually diminishes, this diminution being probably due to the state of inanition, and the consequent diminution of tissue-metamorphosis. Still the quantity is usually in excess of the standard, as long as the fever lasts. In convalescence, it may be, for several days, much below the standard, the destructive metamorphosis of tissue being now checked, and the formative processes being unusually active.

According to Brattler, there is a close correspondence between the amount of urea and the temperature, the greater the amount of urea, the higher the temperature. Although the temperature may be subject to daily variations, according to the amount of evaporation from the skin, and other circumstances, yet taking the whole course of the fever, it is found, that both the temperature and the quantity of urea are greatest in the first week, and afterwards gradually diminish.

An increased amount of urea, however, is not always constant

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[o] NEUBAUER'S *Anleitung*, 2nd ed. p. 248.
[p] See note, page 11. [q] *Gaz. Méd. de Paris*, 1857, p. 193.
[r] See PARKES, *loc. cit.* [s] WARNECKE, 1860. [t] *Op. cit.*
[u] JONES, 1857, 1858 (1).

throughout the fever. There are certain incidental circumstances which may lead to its diminution, even below the standard of health; and this may account for the fact that some observers have thought that the quantity of urea in pythogenic fever is really diminished. It is possible that the quantity may be reduced, owing to the occurrence of some local inflammation. For example, in one case, Dr. Parkes ascertained that the amount of urea during an intercurrent attack of pleurisy, was one-third less than the average amount before the pleurisy. Dr. Parkes, however, has shown that the amount of urea is not influenced by the diarrhœa; and hence, the intestines cannot be regarded as a channel for eliminating urea, in Pythogenic Fever. But again, an increased amount of urea may be formed in the system, but may accumulate in the blood, in place of being excreted by the urine. The cause of this non-elimination is not always apparent; but in most instances it is probably due to an altered condition of the secreting tissue of the kidneys, as shown by the presence of albumen and blood-casts in the urine. The consequences of non-elimination appear to be the same as in Typhus and Relapsing Fever, viz.: the production of delirium, stupor, coma, convulsions, and other symptoms of the typhoid state. It has been found, that a deficiency of the solids of the urine often coincides with the supervention of these symptoms.[x]

The Uric Acid is always increased, and may be more than double the normal standard. Thus the normal daily amount for an adult male, being, according to J. Vogel, 7·72 grains, Dr. Handfield Jones found as much as 23·76 grains, excreted by a man aged 24, on the seventeenth day of the fever.[y] According to Zimmermann,[z] the quantity increases up to the fourteenth day, and then diminishes. As in typhus, deposits of lithates may occur at any stage of pythogenic fever, and are not necessarily critical. During convalescence, the excretion of uric acid, like that of urea, is usually for some days below the normal standard.

Chloride of Sodium, of which about three drachms is passed in the urine daily by the healthy adult, is diminished sometimes to a mere trace. This diminution may be partly due, as suggested by Vogel, to the small quantity taken with the food, and partly to the large amount of chloride of sodium passed with the stools. Parkes, however, has noticed a great diminution occur, without any change of diet, in cases where there was no diarrhœa or pneumonia, so that, as in typhus, there appears to be an absolute reten-

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[x] PARKES, *On Urine*, 1860, p. 252. [y] JONES, 1857. [z] ZIMMERMANN, 1852.

tion of chlorides in the system (see page 147). During convalescence, the chlorides are passed in abundance.

Albumen is less frequently present in the urine than in typhus, and when it occurs, it appears *later* in the disease. I have examined the urine daily in 25 cases, and have found albumen only in 5. Parkes found albumen in 7 of 21 cases; Brattler, in 9 of 23 cases; Martin Solon,[a] in 21 of 54 cases; Becquerel, in 8 of 38 cases;[b] Finger, in 29 of 88 cases;[c] and Friedreich, in 14 out of 33 cases.[d] Adding together these results, albumen was discovered in 93 out of 282 cases; or in 32·26 per cent.[e] The quantity of albumen is usually small, and its duration temporary. The albumen rarely appears before the middle of the third week. In none of my cases was it present before the sixteenth day. In all of Finger's cases, it appeared between the sixteenth and twenty-fifth days. Its appearance coincides with the time that cerebral symptoms usually supervene; and in this respect, its later occurrence than in typhus, is a fact of no small interest. (See page 148.)

Cases of pythogenic fever with albuminuria are usually severe, and have the typhoid state well-developed. Of 50 cases observed by Finger and Martin Solon, 27 died. Kerchensteiner also found albumen chiefly in severe cases. Martin Solon found the kidneys in the fatal cases congested and hypertrophied, and presenting the appearances of incipient Bright's disease. The remarks made at page 149, with regard to the occurrence of albuminuria in typhus, are equally applicable here.

Epithelium, *Blood*, etc. Renal epithelium and tube-casts may, or may not, coexist with albumen, in the urine. Zimmermann says that tube-casts may be found, even when there is no albumen. In 3 severe cases, I have found the urine to contain blood.

Leucine and Tyrosine have been found occasionally in the urine by Frerichs, under the same circumstances as in typhus. (See pp. 149, 195).

Phosphates. In the advanced stages, when the urine is but feebly acid, I have usually found it to deposit a large amount of earthy, and triple phosphate.

2. *Retention and Incontinence of Urine* (see *Symptoms* under *Nervous System.*)

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[a] Solon, 1847. [b] *Brit. & For. Med. Chir. Rev.* Ap. 1852, p. 316.
[c] *Prag. Vierteljahrsch.* 1847, iii. 28. [d] Schmidt's *Jahrb.* 1855, Bd. 86, s. 172.
[e] Some observers seem to have found albuminuria more frequently. Griesinger found it in 31 of 36 cases, and Trotter in all of 20 cases. (Griesinger, 1857; Trotter, 1854).

g. Symptoms referrible to the Nervous and Muscular Systems.

1. *Headache*, as in typhus, is one of the first, and most constant symptoms. I ascertained that it existed in 77 out of 82 cases; and Louis noted it in all but 7 of 133 cases.[f] It is probably as common in children, as in adults.

Of 126 cases, where Louis noted its date of commencement, it existed from the first in 112; and in all the remaining 14, it commenced, on or before the sixth day. It is most severe during the first week; and by the end of the second week, it has usually ceased. I have rarely known it to co-exist with delirium.

The pain is usually confined to the forehead; at other times, it extends over the entire head.

The pain is not usually very intense. In 15 of 67 cases, I have noted it as severe. Even when severe, it is rarely described as of a shooting, or bursting character: it is more commonly dull and heavy.

2. *Vertigo* is often present from the first, and it may persist throughout the disease. It was complained of by 36 out of 55 patients, whom I questioned on the point.

3. *Pains in the Back and Limbs*, of an aching or undefinable character, are rarely absent from the commencement. They are most severe in the lower extremities. The pain in the back never approaches, in severity, that of small-pox. In several instances, I have known the pains in the limbs, of a neuralgic character, and so severe, as to prevent sleep. (See page 150.)

4. *Impairment of the Mental Faculties. Delirium.* Pythogenic fever contrasts strongly with typhus, in the circumstance, that a large proportion of the patients pass through the attack, without any delirium, or impairment of the mental faculties. Out of 100 cases, I ascertained that the intelligence remained perfectly clear throughout the attack in 33; a few of the patients may have talked or moaned a little in their sleep; but when awake, they appeared as rational as in health. Moreover, 3 of the 33 patients died; 2 from perforation of the bowel, and 1 from epistaxis. Louis appears to have made a similar observation, for in 32 out of 134 of his cases, there was neither somnolence nor delirium. Moreover, 8 of his cases, in which there was no delirium, were fatal; 6 of them, from perforation.[g] In 2 of Jenner's 23 fatal cases, there was no delirium or mental confusion.[h]

In 67 of 100 cases, I ascertained that there was either delirium or mental confusion; but in many of these cases the delirium was only slight and occasional, it occurred chiefly in the night-time, and the patient was at most times quite rational. Of the 67 cases, 18 were

[f] Louis, 1841, ii. 1. [g] Ibid. ii. 18. [h] Jenner, 1849 (2).

fatal. In only 22 cases, of which 11 were fatal, was there at any time complete unconsciousness.

The character of the delirium varies, as in typhus (see page 153). At first, it is often active and noisy, the patient screaming and shouting, and being, with difficulty, kept in bed. As he becomes more prostrate, this active delirium may pass into the forms of typhomania and delirium tremens described under the head of typhus. The more active and noisy the delirium, the greater is the danger. Of 17 cases, in which Louis noted active delirium, 12 died; and of 18 of my cases, where it occurred, 9 were fatal. My observations confirm those of Dr. Jenner, to the effect, that this form of delirium is more common than in typhus, although the fact is probably, in part, accounted for by the circumstance, that the patients are usually younger and more robust, before the attack, than in typhus. (See page 154.)

The delirium is later in making its appearance than in typhus. As a rule, it does not commence until the middle or end of the second week; and it often does not appear until the end of the third week, and then it only lasts for a day or two before death or recovery. Still, there are exceptional cases, where the delirium occurs earlier. In 9 out of 100 cases, I have noted delirium as occurring during the first week; and, in one instance, I have known violent delirium on the second day of the attack (see also page 439). Bristowe has recorded a case, where maniacal delirium occurred on the second day.[h] Louis mentions two instances where the patients were delirious during the first night.[i] Jenner observes, that active delirium is occasionally one of the earliest symptoms, and may then cease on the appearance of the eruption.[k]

The delirium is almost invariably greatest in the night-time, and sometimes it occurs only in the night.

The delusions, under which the patient labours, are similar to those of typhus (see page 154). When the delirium is active, the mind appears to wander from one subject to another; but in the quieter forms, it is usually centred on some fixed object. Chomel mentions a case, where the patient continually demanded to be bled.[l] One of Louis's patients could not be persuaded that he had not been robbed; another insisted that, during his illness, he had been to his native village, and had brought back some wolf's cubs, which he wanted to sell. In one of my cases, the patient's ideas fluctuated

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[h] *Trans. Path. Soc.* Jan. 7th, 1862.
[i] Louis, 1841, ii. 32.
[k] Jenner, 1853, p. 234.
[l] Chomel, 1834.

between money and his mother; and, in another, the patient always burst out laughing when spoken to. Some patients, in the midst of the most dangerous symptoms, insist that there is nothing the matter with them, a form of delirium which, in Louis's experience, always terminated fatally.

In children, delirium presents much the same characters as in adults. Taupin noted acute delirium in 44 out of 118 cases. Rilliet only observed delirium in one-third of his cases.[m] It commences, on the whole, rather earlier than in adults, but rarely before the second week.

5. *Wakefulness, Somnolence, and Coma.* During the first week or ten days of the disease, the patient is usually wakeful at night, and the sleep is much disturbed. I have noted these symptoms in 76 out of 100 cases. On the whole, however, the wakefulness is less complete than it often is, in the early stage of typhus.

But in many cases, about the middle or end of the second week, a certain amount of somnolence supervenes. This I have noted in 57 out of 100 cases. Louis observed somnolence in 102 out of 134 cases: of his 46 fatal cases, it was absent only in 5. In severe cases, somnolence is sometimes observed at an early stage. Of Louis's 46 fatal cases, it was observed on the first day in 4, and during the first week, in 5 others; whereas, of 88 cases which recovered, it was observed, during the first week, in only 2.[n]

At first, the somnolence is slight, and the patient is easily roused; but, in severe cases, it gradually becomes more profound. Complete unconsciousness, however, is rarer than in typhus; it occurred in 22 of my 100 cases. Occasionally, the patient lies for several days perfectly still and quiet, and appears to understand everything that is said and done, but is unable to articulate intelligible replies. The dilated pupils, half-closed eyes, and languid, apathetic, rather than stupid, countenance, accompanying this condition, might cause the case to be mistaken for hysteric coma. Dr. W. T. Gairdner has called attention to this condition;[o] and I have met with many cases, answering more or less to his description. In one instance, where the patient had been in this state for upwards of a week, although occasionally delirious, she bade adieu to her friends an hour before her death, apparently quite aware of her approaching end. I have never observed any state approaching to this, in typhus.

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[m] Louis, 1841, ii. 35. [n] Ibid. p. 6. [o] Gairdner, 1862 (2), 144.

The somnolence is often interrupted by delirium. During the night, the patient is wakeful and delirious, and in the day-time stupid and drowsy; but in severe cases, the somnolence gradually becomes more constant, and lasts until death, or the commencement of convalescence. More or less somnolence, in most cases, precedes delirium;[p] whereas, in typhus, the patient is usually wakeful and delirious before he becomes drowsy.

True coma-vigil (see page 157) rarely, if ever, occurs in pythogenic fever.

Somnolence follows the same rule in children, as in adults. West has known it so overwhelming, at the outset of the disease, that the child fell asleep two or three times during breakfast.[q]

6. *Prostration.* In all cases of pythogenic fever, there is some muscular prostration from the first, which increases with the progress of the disease; but, as a rule, the degree of prostration is much less than in typhus. (See page 157.) A large proportion of patients (44 out of 100 of my cases) are able to sit up, and get up to stool, throughout the attack; and I have repeatedly known persons, who had the disease in a mild form, attend from first to last, as out-patients at a hospital. Even in fatal cases, there may be comparatively little prostration, up to the time of death. I have often known patients get up to stool, within twenty-four hours of the fatal event; and one of my patients, who walked a long distance to hospital, died thirty-six hours after admission. Louis mentions similar cases;[r] and Jenner says that, in two of his 23 fatal cases, the patients were able to leave their beds unassisted and with facility, throughout the disease.[s]

Complete prostration (which occurred in about one-third of my cases) appears later than in typhus. It is rare before the middle of the third week.

Out of 62 patients, I ascertained that only 7 kept their bed from the first; while 21, or more than one-third, did not take to bed until after the first week; and 6 continued going about until the third week. Again, of 94 patients, only 6 were admitted into hospital before the fifth day; and, in most of the 6, unusual attention had been directed to the disease, from the occurrence of other cases in the same family. Eighty of the patients, or 85 per cent., had been ill more than five days on admission; 59, or 63 per cent., more than seven days; and 31, or 33 per cent., fourteen

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[p] 'Le délire *débutait*, chez presque tous les sujets, après la somnolence.' Louis, 1841, ii. 20.

[q] West, 1848, ed. 1854, p. 558.

[r] Louis, 1841, ii. 67.

[s] Jenner, 1849 (2).

days or upwards. The mean duration of all the cases, prior to admission, was 11¼ days. Contrast with these results, those obtained in the case of typhus. (See page 158.)

7. *The Decubitus,* as in typhus, is usually dorsal. (See page 158.)

8. *Muscular Paralysis.* The stools and urine are passed involuntarily, in most cases where there is complete prostration; but, on the whole, these phenomena are rarer than in typhus. I noted them in only 21 of 100 cases; retention of urine, requiring the catheter, existed in only 2 cases. Involuntary evacuations were noted by Jenner in only 10 of 23 fatal cases, and retention of urine only once. (See page 159.)

Inability to protrude the tongue, and dysphagia, are occasionally observed in grave cases; but, as already stated, dysphagia may depend on other causes than paralysis, as does also the tympanitis. Other forms of paralysis will be alluded to, under the head of 'Complications.'

9. *Muscular Agitation.* Tremors of the hands or tongue, or quivering of the lips, I have noted in 27 out of 100 cases; 8 of the 27 cases were fatal, and 13 of the remaining 73 cases. In 11 cases, of which 6 were fatal, the tremulousness was very marked. These tremors may occur in young persons, who have never been addicted to spirits (see page 159). They are also sometimes observed in cases where there is no delirium, and where the intelligence is perfectly clear. Severe tremors, unaccompanied by much mental disturbance, according to Jenner, often accompany deep ulceration and sloughing of Peyer's patches.

Spasmodic movements, such as subsultus, twitchings of the mouth, carphology, and protracted hiccup, are only observed in the advanced stage of severe cases. I noted them in 11 of 100 cases; 8 of the 11 cases, but only 13 of the remaining 89 cases, were fatal. In 1 case, subsultus lasted for five days before the fatal event. Louis observed these symptoms in 18 out of 134 cases; 12 of the 18 cases were fatal, but only 34 of the remaining 116 cases.[t] Jenner noted subsultus in 6, and carphology in 2, out of 21 fatal cases.[u]

In children these symptoms are probably less frequent, but they are of equally grave signification. Barthez and Rilliett noted carphology 7 times, and subsultus 4 times (probably in the same cases) out of 107 cases.[x] They also allude to two cases, where choreic

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[t] Louis, 1841, ii. 44.

[u] Jenner, 1849, (2).

[x] Barthez and Rilliet, 1853, ii. 681.

movements occurred in the course of the fever. According to West, 'even when the disease is severe, neither subsultus nor 'floccitation is frequent.'[y] It is difficult to understand M. Taupin's statement, that carphology occurs 'dans presque tous les cas,' and that he observed subsultus in 79 out of 121 cases, except on the supposition that he attached a different meaning to these terms from other writers. In most severe cases, however, children pick their nose or lips, until they bleed.

10. *Muscular Rigidity.* Rigid contraction of the muscles of the extremities, or of the neck, was observed by Louis in 4 out of 134 cases; and rigidity of the trunk by Rilliet and Barthez, in 5 out of 107 children; all 9 cases were fatal. Chomel,[z] Barth,[a] and Jackson,[b] however, each record one example, where the patient recovered. Barth also observed a case of cataleptic rigidity which recovered; and two similar cases, both females, have occurred in my own practice.

11. *General Convulsions* are rarer than in typhus, but are equally formidable. They occurred in two of the cases analysed in this work. In one of the cases, convulsions came on, after much delirium, on the sixteenth day: and the patient died comatose, half an hour afterwards. The urine was albuminous; and the cortical substance of the kidneys was found to be much hypertrophied, and pale; the renal epithelium was loaded with oil, so that the disease was probably of some standing. In the second case, general convulsions, with foaming at the mouth, and biting of the tongue, occurred as early as the eleventh day. The fit was preceded by acute delirium, and followed by much drowsiness; but the patient ultimately recovered. The urine, on the day of the convulsions, did not exceed 12 ounces, and its specific gravity was 1012; but it contained no albumen. In one other instance, I have known a patient recover after a severe fit of convulsions, which occurred on the fourteenth day, and lasted for a quarter of an hour. For several days before, there had been great restlessness and delirium; but the fit was immediately preceded and followed by stupor. The urine, unfortunately, was not examined. The bearing of these observations will be understood by referring to page 162.

Barthez and Rilliet mention 5 instances of convulsions occurring in children; 4 of the 5 died; the condition of the urine and kidneys is not stated.[c] According to West, convulsions, followed

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[y] West, 1848, ed. 1854, p. 560. [z] Chomel, 1834, [a] Louis, 1841, ii. 63.
[b] Bartlett, 1856, p. 54. [c] Barthez and Rilliet, 1853, ii. 682.

by coma, constitute not an uncommon mode of fatal termination in children.[d]

h. Morbid Phenomena presented by the Organs of Special Sense.

1. *Organs of Vision.* Increased vascularity of the conjunctivæ is comparatively rare; and, when it occurs, it is much later in appearing than in typhus, and the blood, in the conjunctival vessels, is of a brighter hue. I have noted great vascularity of the conjunctivæ, in 8 out of 100 cases, and pain in the eyes, without increased vascularity, twice. I am not positive that these symptoms were absent in all of the remaining cases; but, I am persuaded, that they were much less frequent than in the cases observed by Louis, who noted increased vascularity in 38 out of 60 cases; and tenderness, without vascularity, seven times.[e] Of 13 fatal cases observed by Jenner, the conjunctivæ were pale in 10, and injected in only 3.[f] Bartlett also observes that congestion of the conjunctivæ is rare.[g] (See page 169.)

In 6 cases I have known the eyelids kept closed in the advanced stages, as if from intolerance of light: 3 of the 6 died. Louis says that, in 4 fatal cases, he had known the eyelids so firmly closed, that it was difficult to force them open; and that he had never known a patient, presenting this symptom, recover. He was inclined to regard it as of a purely spasmodic character, analogous to the permanent rigidity occasionally observed in the arms and neck. In one instance, this firm contraction of the eyelids lasted for upwards of a fortnight.[h]

Dr. Jenner was the first to point out the dilated condition of the pupil in pythogenic fever, as contrasted with the small pupil of typhus. Of 23 fatal cases, he observed the pupils dilated in 7, and contracted in 2.[i] In fully three-fourths of my cases, the pupils were abnormally dilated, at some stage of the fever, and Dr. W. T. Gairdner has made similar observations at Edinburgh.[k] Dilatation of the pupil may be observed after the tenth day, in cases where there is no delirium, or impairment of the mental faculties, or it may co-exist with delirium, and especially with that condition approaching to hysteric coma, already described (see page 489). In cases, however, where there is great stupor, and complete unconsciousness, the pupils are often contracted, and I have then often known them to be as contracted as in any case of typhus. (See page 169.)

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[d] West, 1848, ed. 1854, p. 561.
[e] Louis, 1841, ii. 87. [f] Jenner, 1849 (2). [g] Bartlett, 1856, p. 53.
[h] Louis, 1841, ii, 88. [i] Jenner, 1849 (2). [k] Gairdner, 1862 (2), 148.

Some patients complain of haziness of vision, increased by sitting up. In rare cases, strabismus is observed.

2. *Organs of Hearing.* Ringing and buzzing sounds in the ears are often complained of, in the early stage of the disease. Louis noted them in 36 out of 99 cases, and Barth in 85 out of 129 cases.[l] According to Louis, they are more severe, and last longer, in the severe cases, than in the mild.

Deafness, of one or both ears, is a common symptom. I noted it in 20 out of 46 cases; Louis observed it in 58 of 99 cases; Barth, in 36 of 129 cases; and Jenner, in 6 of 23 fatal cases. It is rarely observed before the end of the second week. As Louis observes, 'La plus extrême surdité n'ajoute rien à la gravité 'du pronostic.' Trousseau draws a distinction between deafness of one, or of both ears. Deafness of one ear he thinks unfavourable, as it is apt to arise from suppuration of the ear, which may excite meningitis. On the other hand, he asserts that he has scarcely ever known a patient die, after having deafness of both ears, which he attributes to catarrh of the eustachian tubes.[m] My experience does not lead me to regard deafness in quite so favourable a light, and, probably, the remarks made at page 170, are equally applicable here.

3. *Cutaneous Sensibility.* In 2 out of 40 cases, I have noted the existence of great hyperæsthesia of the entire cutaneous surface. One of the patients was a female, aged 20; the symptom first showed itself on the seventeenth day, and lasted three days. The second patient was a female aged 18, and the hyperæsthesia was first noticed at the commencement of convalescence. On the other hand, Rilliet and Barthez speak of anæsthesia as an occasional grave symptom in children.

4. *Epistaxis* is a common symptom, but appears to be more frequent at Paris than elsewhere. Thus, while Louis and Barth found it in 91 of 156 cases,[n] Dr. Flint noticed it in only 21 of 73 cases in America;[o] Jenner, in 5 of 15 fatal cases; and it occurred in only 13 of 58 cases noted by myself. As to children, Rilliet and Barthez speak of epistaxis as occurring in one-fifth of their 107 cases,[p] and Taupin observed it in only 3 of 121 cases.[q]

The hæmorrhage may take place at any period of the fever, and may recur repeatedly. The quantity of blood lost may vary from a few drops to many ounces. All observers agree in stating, that the bleeding is never followed by any relief to the symptoms;

[l] Louis, 1841, ii., 93. [m] Trousseau, 1861, p. 170.
[n] Louis, 1841, ii., 84. [o] Flint, 1852.
[p] Barthez and Rilliet, 1853, ii. 685. [q] Taupin, 1839.

while, on the other hand, it may be so profuse, as to be the immediate cause of death. Three examples of death from epistaxis have come under my notice.

Case XXXII.

Pythogenic Fever.—Death on the 10th day from Epistaxis.—Autopsy:—Enlargement of Spleen and Mesenteric Glands.—Commencing Ulceration of Peyer's Patches.

Mary F——, aged 20, a servant in a gentleman's family, was admitted into the Fever Hospital on July 29th, 1857. Was taken ill on the 22nd, at Ramsgate, where she had been on a visit for three weeks. Her symptoms, before admission, had been cold shivers, headache, pains in the limbs, urgent diarrhœa, and prostration.

July 30th (eighth day). Pulse 120. Slept well; is free from pain, and intelligence clear. Skin hot and dry; face much flushed; flush circumscribed; one or two lenticular spots; tongue furred and red at edges; abdomen tympanitic; gurgling, but no tenderness, in right iliac fossa; three watery stools. Was ordered beef-tea and milk, a starch and opium enema, and a mixture containing acetate of lead (gr.; iij.) and liq. morph. acet. (mlij.) after each motion.

August 1st (tenth day). No worse until 9 p.m., when she began to bleed from the nose very profusely. When seen about an hour after, the pulse was almost imperceptible, the skin cold, and the features pinched. Four stools to-day, but no blood in any of them. Cold was applied to the forehead, and 10 grains of gallic acid, with 20 minims of sulphuric acid, were ordered every hour. The bleeding, however, continued, and the patient died at 11.40 p.m., before plugging could be resorted to.

Autopsy, 31 hours after death.

Cadaveric rigidity well-marked.

All the internal organs very pale and anæmic; old adhesions over left lung.

Liver 34 ounces, very pale. A little pale, thin bile in gall-bladder. Spleen 9 ounces; very soft. Mesenteric glands much enlarged, one or two almost as large as walnuts; their surface on section much injected.

The stomach and upper part of the small intestines contained several ounces of coagulated, and of fluid dark blood, but the mucous membrane of this portion of the digestive canal was healthy. There was no blood in the lower portion of the bowel, which was diseased. About a yard above the cæcum, Peyer's patches began to be diseased. The number and extent of the diseased patches increased towards the cæcum: many of the patches were elevated fully one-eighth of an inch above the surface, and contained a cheesy, yellow deposit. The mucous membrane over most of them was intact, but on one or two, close to the ileo-colic valve, there was slight ulceration. The mucous membrane, between the patches, was intensely

injected. The solitary glands, in the cæcum, ascending colon, and lower part of the ileum, were likewise elevated, and contained a cheesy deposit.

i. Emaciation.

In cases of pythogenic fever, protracted to three or four weeks, there is usually great, and often extreme, emaciation. The difference from typhus, in this respect, is very remarkable. (See page 228.)

Section VII.—Stages and Duration.—Relapses.

Although any sub-division of pythogenic fever into distinct stages, must be, in a measure, artificial, the following stages may be observed in many cases; 1, the stage of incubation; 2, the stage of invasion; 3, the stage of reaction; 4, the nervous stage; 5, the typhoid stage; 6, the stage of lysis; and 7, convalescence. The duration of these stages varies in every case; and the second and third are often wanting.

1. *The stage of Incubation* is considered at page 435.

2. *The stage of Invasion* lasts for one or more days, and extends from the first feeling of illness, until the development of decided febrile symptoms.

The invasion is often so gradual, that neither the patient nor his friends can state the precise day on which the illness commenced. This was the case with more than one-half of 63 patients under my care. Jenner could only ascertain the day of commencement in 7 of 15 fatal cases.[p] Louis and Chomel[q] speak of the invasion as being in most cases sudden; but the experience of Forget, as well as of Bartlett, and of most American writers confirms that of Jenner and myself. At all events, the contrast, which pythogenic fever presents, in this respect, to typhus and relapsing fever, is remarkable. (See pages 172, and 349.)

Of 63 cases, where I have noted the mode of commencement, pains in the head and limbs were among the earliest symptoms in 56: and most of these patients also suffered from irregular chills, languor and giddiness; in only 3 cases, did the disease commence with anything approaching to rigors. In 12 cases, there was great nausea and vomiting; in 5, considerable pain in the abdomen; and in 26, or 41 per cent., diarrhœa. In several of these last cases, *the patients had been suffering for a week or two from ordinary autumnal diarrhœa, before any symptom of fever appeared.* This mode of commencement is, I believe, far from uncommon. (See page 455.)

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[p] Jenner, 1849 (2). [q] Louis, 1841, i. 419; Chomel, 1834.

In several cases, also, I have seen the disease, during the first few days, present all the phenomena of ague.

3. *The stage of Reaction* extends from the second or third day to about the tenth or fourteenth day, and is characterized by fever of an irregularly remittent character, headache, vertigo, disturbed sleep, copious excretion of urea, red and furred tongue, tympanitis, diarrhœa, and occasionally vomiting and epistaxis. During this stage, the eruption appears. In many cases, this stage is continued until convalescence.

4. *The Nervous Stage* is characterized by somnolence interrupted by acute noisy delirium, or by a condition approaching to hysteric coma, (see page 489), by dilated pupils, deafness, dry and brownish, or red, glazed and fissured tongue; membranous flakes, and often blood, in the stools; and febrile symptoms of a more continued character, than in the stage of reaction. This stage may commence about the tenth or fourteenth day, or not until the end of the third week: it lasts, until it is succeeded by one of the two following stages.

5. *The Typhoid, Putrid, or Malignant Stage* is characterized by all the phenomena of the typhoid stage of typhus, viz., great prostration, stupor passing into coma, low muttering delirium, dry brown tongue, tremors, subsultus, and involuntary evacuations. (See page 173.)

Louis proved to demonstration,[r] and all pathologists now admit, that the cerebral symptoms of pythogenic fever are independent of inflammation of the brain, or of its membranes. Of late years, it has been the custom to refer them to *septicæmia*, produced by the absorption of pus, or putrid matter, from the intestinal ulcers.[s] Admitting that such an absorption is possible, it appears to me doubtful, if it accounts for the phenomena, which have been ascribed to it. There is no relation whatever between the presence and severity of cerebral symptoms, and the extent of the intestinal disease. I have known death preceded for a week by the typhoid state, although only two small ulcers existed in the bowel. Again, in rare instances, cerebral symptoms, and even the typhoid state, may occur, before ulceration has commenced, while all the phenomena of the typhoid state are developed in many other diseases, in which there is no ulcerated surface from which pus can be absorbed.

It is highly probable, that the phenomena of the typhoid state, are due, as in other diseases (see page 174), to the retention in

[r] Louis, 1841, ii. 22.

[s] Piorry applied to pythogenic fever, the designation *Entérite Septicémique*. See also Todd, 1860, p. 113; Gairdner, 1862 (2), p. 200.

the blood of those products of tissue-metamorphosis, which ought to be eliminated by the kidneys. On the supervention of the typhoid state, it is found that the urinary solids, which have previously been so much in excess of the normal amount, diminish (see page 485); and in several instances, I have ascertained, that the quantity of urea excreted in twenty-four hours, diminished on the advent of cerebral symptoms, and increased again, on their cessation. In one case, the quantity which was 292 grains, when the patient was delirious and unconscious, rose to 964 grains when the delirium abated, and the consciousness returned. In another case, the quantity, which at first was 422 grains, fell to 352 grains, on the appearance of delirium and stupor; and rose to 490 grains, when these symptoms ceased (see Cases XXVI. and XXX).[t]

6. *Stage of Lysis.* The termination of pythogenic fever, like its commencement, is always gradual. It is not marked by any critical evacuation, and it is usually difficult to say, when convalescence really commences. The resolution of the fever takes place by what has been termed *Lysis*. There is no sudden fall of the pulse, or cessation of the symptoms indicative of danger, which we so often observe in typhus. Moreover, in not a few cases, all the more urgent symptoms undergo amelioration, the tongue becomes cleaner and moister, the diarrhœa and cerebral symptoms abate; but fresh lenticular spots may continue to show themselves, and as long as this is the case, the patient is liable, at any time, to a sudden aggravation of the disease.

7. *Convalescence,* even when fairly commenced, makes slower progress than in typhus, a circumstance accounted for by the greater emaciation, and by the existence of the uncicatrized ulcers in the bowel. It is also very liable to be interrupted by relapses, exhausting diarrhœa, peritonitis from perforation, and other dangerous sequelæ, mentioned under the next section. Even when no complications exist, the pulse is sometimes quicker in convalescence, than during the fever. (See page 476.)

Duration.

The ordinary duration of pythogenic fever is from three to four weeks. Of 75 cases which recovered, and in which I was able to determine the point with tolerable certainty, the duration was:— 12 days in 1 case; from 14 to 21 days, in 19; from 22 to 28, in 40; 29 or 30 days, in 9; and above 30, in 6 cases.

Thus, in nearly three-fourths of the total number, the duration

[t] The experiments were performed, some years ago, by Dr. Sanderson and myself, without reference to any connection between the amount of urea and cerebral symptoms.

exceeded three weeks; and in one-fifth, it was more than four weeks. The mean duration of the 75 cases was 24·6 days. The mean duration of 12 other cases, which were fatal, was 22·08 days.[u] The mean duration of 11 fatal cases, noted by Jenner, was 22 days.[x] These results are in accordance with the thermometric observations of Zimmermann, who found that the temperature fell sometimes between the 21st and 28th day, and that, at the same time, the body began to gain in weight. It is obvious, that pythogenic fever, even apart from complications and the chances of a relapse, is a much more protracted disease than typhus. (See page 177.)

At the same time, the fever occasionally terminates, either in death or in recovery, at an earlier date than might be inferred from the above data. It may terminate favourably in a week or ten days, and then, if there be no eruption, the case is regarded as an example of febricula or of mild remittent fever, although the circumstance of other persons in the same family having well-marked pythogenic fever, occasionally demonstrates its real nature. In one instance, I have known convalescence commence about the twelfth day, although the eruption had been distinct (Case XXXIII.). In such cases, it is probable, that the deposit in the intestinal glands is absorbed, and that ulceration never takes place. Again, in Case XXVII., the patient died on the sixth day of the disease. Bretonneau,[y] Forget,[z] Jenner,[a] and Bristowe,[b] each record a case, fatal on the fifth day; Trousseau gives the details of a case which was fatal in less than four days;[c] and cases have been already referred to, where a fatal termination took place on the second, or even on the first, day. (See pages 438-9.)

On the other hand, the fever may be more protracted than is perhaps commonly believed. As long as fresh spots continue to appear, it cannot be regarded as having terminated. Dr. Jenner has expressed the opinion that, except in cases of relapse, fresh spots never appear after the thirtieth day, and that when the febrile symptoms are protracted beyond that date, they are always due to some incidental complication. Although I believe that this statement, as a rule, holds good, I have met with several instances in which fresh spots continued to appear as late as the thirty-fifth day; and in one remarkable case, where the general symptoms were mild, fresh spots were noted almost daily, from the

[u] Cases are not included in these calculations, in which there was a relapse, or in which the illness was prolonged by complications, after the spots had ceased to appear on the skin.

[x] JENNER, 1849 (2).

[y] BRETONNEAU, 1829, p. 70.

[z] FORGET, 1841, p. 119.

[a] JENNER, 1853, p. 260.

[b] *Lancet*, Ap. 28th, 1860, p. 422.

[c] TROUSSEAU, 1861, p. 168.

14th to the 60th day (Case XXXIV.) In all of the cases referred to, there was no difficulty in determining the date of commencement of the disease, within a day or two.

My observations lend no support to the doctrine of critical days, as applied to pythogenic fever; but I have often noticed that it terminated on, or about, the 21st or the 28th day.

Case XXXIII.

Pythogenic Fever of a mild form. Convalescence on the thirteenth day.

Ellen T——, aged 22, was admitted into the London Fever Hospital, November 17th, 1857. She had been taken ill on November 10th, and had kept her bed for three days. Her illness commenced with pains in the limbs, giddiness, nausea and vomiting, loss of appetite, and great thirst. For four days before admission, the bowels had been open four or five times a day.

November 17th (eighth day). Pulse 100. Has much giddiness, and slight headache, and slept badly, but intelligence is clear, and expression is natural. Pupils rather large; skin warm; faint pink flush on cheeks; several lenticular rose-spots on chest and back. Tongue moist and furred, red at edges. Purging has ceased. Great thirst.

November 21st (twelfth day). Pulse 94. Feels better. Sleeps well at night. Several fresh spots have appeared daily since admission, and on November 18th had slight epistaxis. Tongue cleaner; thirst abated; bowels not opened for two days.

November 22nd. Pulse 96. Spots have all disappeared, and appetite is returning.

November 24th. Pulse 80, and is able to get up without assistance.

The patient continued to gain strength daily, and was discharged on December 12th.

Case XXXIV.

Pythogenic Fever, remarkable for its long Duration.

William S——, aged 20, was admitted into the London Fever Hospital on July 9th, 1858. His illness commenced on June 27th, with diarrhœa, cold shivers, and pains in the limbs.

July 10th (fourteenth day). Pulse 96. Some quiet delirium in the night. Circumscribed flush on cheeks, and about twenty lenticular rose-spots on chest and abdomen. Tongue moist and furred; abdomen tympanitic and tender; four light watery stools.

Fresh spots were noted almost daily from this date till August 25th, and on no day were they entirely absent. The pulse varied from 96 to 132. The tongue, for a few days, was dry and brown; but after July 26th, it was moist, red, and fissured. The bowels were all along relaxed; scarcely a day passed, that the patient did not pass from two to six light watery stools. On August 9th, there was considerable epistaxis. The

intelligence was always good, and after July 20th the delirium at night ceased. The pupils were mostly dilated, and from August 9th to 20th, there was considerable deafness. The appetite began to return on August 3rd, and on August 14th the patient was very hungry, although the pulse was 120. Twenty lenticular spots were counted on the body, and there had been four light watery stools. The patient ultimately recovered, and was discharged on September 10th.

Relapses.

True relapses are occasionally observed, during convalescence from pythogenic fever. They occurred in 10 out of 142 cases, of which I have notes. Peacock met with them in 6 out of 35 cases.

After a convalescence from the first attack, of about ten days or a fortnight, the patient is seized with chilliness or shivering, headache, and often sickness, followed by a return of all the former symptoms, including diarrhœa, re-enlargement of the spleen, and a fresh eruption of lenticular spots. The eruption usually appears on the fourth or fifth day of the relapse; but, in some cases, it is seen as early as the second day, and in others not until the eleventh; the spots may be more abundant than in the primary fever. Wunderlich[d] and Thierfelder[e] have noted, with the thermometer, a similar increase of the temperature, to what is observed in the first attack.

The duration of the second attack is usually shorter than that of the first. Of 24 cases collected from various sources by Michel,[f] the mean duration of the first attack was 27 days; of the intermission, 11 days (shortest 2, and longest 31 days); and of the relapse 16 days (longest 30). In ten cases, which have come under my own observation, the duration has been as follows:—

Age.	Sex.	1st Attack.	Intermission.	Relapse.	Total Duration.
25	M.	37 days.	10 days.	14 days.	61 days.
15	F.	26 „	14 „	11 „	51 „
16	F.	36 „	16 „	13 „	65 „
5	M.	22 „	14 „	10 „	46 „
14	M.	32 „	9 „	10 „	51 „
14	M.	21 „	14 „	16 „	51 „
11	M.	21 „	14 „	14 „	49 „
44	M.	34 „	15 „	10 „	59 „
19	F.	24 „	14 „	16 „	54 „
?	F.	14 „	10 „	12 „	36 „
Average..		26 days.	13 days.	12·6 days.	52·3 days.

Sometimes, the relapse is milder than the primary attack, but in my experience the contrary rule has usually obtained. In two

[d] Wunderlich, 1858. [e] Thierfelder, 1855. [f] Michel, 1859.

cases I have known great delirium in the relapse, where there had been none in the first attack; and in two other cases, there was watery diarrhœa in the relapse only. In one of the two last cases, the mildness of the symptoms and the constipation, which characterized the primary attack, would have rendered the diagnosis doubtful, had it not been for the presence of lenticular spots. In several recorded cases, the pulse has attained a greater frequency in the relapse, than in the first attack, and has been dicrotous in the relapse only. Still the relapse rarely proves fatal, except from some incidental complication. Of my own 10 cases, only 1 died, and there the fatal event was due to an abortion. Of 6 cases observed by Thierfelder, 1 died.

Post-mortem examination of fatal cases discloses the recent intestinal ulcers of the relapse, co-existing with the cicatrices of the first attack; but, as those glands only are attacked, which have formerly escaped, the recent lesions are usually less extensive than after death in ordinary cases. There is also a fresh enlargement of the mesenteric glands, and of the spleen. Trousseau denies that any fresh disease of the bowel occurs in these cases; and, regarding the intestinal lesion, as the specific eruption of the disease, he contends, that they are not true relapses.[g] But in this opinion Trousseau stands alone. The observations of Stewart,[h] Griesinger,[i] Thierfelder,[k] Wunderlich,[l] Peacock,[m] and H. Weber,[n] agree entirely with my own, as above detailed.

Stewart and Trousseau have recorded instances, in which there appears to have been a second relapse, or a third attack; but cases of this sort are extremely rare.

It is difficult to account for these relapses. Age and sex have no influence in their production. The youngest of my patients was 5 years; the oldest 44. Barthez and Rilliet observed relapses in 3 out of 111 children.[o] It is customary to attribute them to errors of diet; but I have never been able to trace them to any such cause, and Thierfelder makes a similar observation. They vary in frequency at different times, and are, I think, most common in autumn. Although they are comparatively rare, I have known three members of one family suffer from them.

Section VIII.—Complications and Sequelæ.

Many of the complications and sequelæ of pythogenic fever

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[g] Trousseau, 1860, p. 158. 'Quoique l'appareil symptomatique soit très 'complet, quoique l'éruption cutanée se reproduise, la lésion caractéristique de 'l'intestin ne se renouvelle pas.'

[h] Stewart, 1840, p. 301.

[i] Griesinger, 1857, p. 178.

[k] Thierfelder, 1855, p. 216.

[l] Wunderlich, 1858, p. 294.

[m] *Trans. Path. Soc.* ix. 209.

[n] Ib. xii. 96.

[o] Barthez and Rilliet, 1853, ii. 691.

are the same as those already described under the head of typhus; others are peculiar to pythogenic fever. The latter only require a detailed consideration.

a. Diseases of the Respiratory Organs (see page 182).

1. *Bronchitis.* Catarrh of the bronchial tubes is, on the whole, less common than in typhus. Still it was so severe as to be noted in 21 of the 100 cases under my care, of which the symptoms have been already analysed, and in several of the cases it was observed early in the disease. (See page 182.)

2. *Hypostatic Consolidation* is much rarer than in typhus, but may be developed, in those cases, where the disease passes into the typhoid state. (See *Anatomical Lesions*, and also page 183.)

3. *Pneumonia*, on the other hand, is more common than in typhus (Cases XXVIII. and XXIX.) It occurred in 13 of my 100 cases, and Flint noted it in 12 of 73 cases.[p] In many cases, indeed, pneumonia is the immediate cause of death. (See *Anatomical Lesions*, and page 184.)

4. *Gangrene of the Lung.* I have met with one or two cases where pneumonia terminated in gangrene; but this complication is very rare.

5. *Pleurisy* is more common than in typhus; and, in rare cases, terminates in empyema, or in the formation of an interlobar pleural abscess. Dr. Peacock records a remarkable case, where a patient, convalescent from pythogenic fever, suddenly expectorated large quantities of pus, and eventually recovered. The pus was thought to be derived from a circumscribed pleural abscess, bursting into the lung.[q]

6. *Tubercle.* In my experience, acute tuberculosis of the lungs is a far more common complication or sequela of pythogenic fever, than of typhus; and it is intelligible why this is the case, when we recollect the more protracted duration of the former malady, and the greater emaciation which it entails. Louis records four fatal cases of pythogenic fever, in which the lungs were found studded with recent tubercles. Bartlett also observes, that consumption is a common sequela of 'typhoid fever' in America.[r] Tubercle ought always to be suspected, when febrile symptoms, with bronchitis and emaciation, persist after the cessation of the primary fever. (See pages 185 and 422.)

7. *Laryngitis* is an occasional, and sometimes a very serious, complication. It may assume different forms. There may be œdema glottidis, or there may be an erysipelatous condition of the

[p] Bartlett, 1856, p. 47. [q] *Med. Times & Gaz.* April 26th, 1862.
[r] Bartlett, 1856, p. 120.

mucous membrane of the larynx, or small collections of matter may form in the sub-mucous tissue; at other times, the mucous membrane becomes coated with diphtheritic exudation, or it is the seat of ulceration. Jenner has recorded two cases of œdema glottidis, in which there was also erysipelas of the head and face; and in one instance, where there was no erysipelas, I have known it cause sudden death; several other cases are recorded by Trousseau.[s] One example of pythogenic fever complicated with diphtheria has come under my notice; Louis records three, and Forget, two, cases; and Barthez and Rilliet mention six cases, where it occurred in children. Ulceration of the mucous membrane of the larynx and trachea constitutes the *laryngitis typhosa* of Rokitansky and other continental pathologists. Louis records two cases, and also a third case, where aphonia, probably due to this cause, lasted for a month. This complication, although apparently more common on the Continent, is occasionally met with in this country:[t] a few examples have occurred at the London Fever Hospital. It is occasionally found unexpectedly after death, in cases where there have been no symptoms referrible to the larynx. In some cases, however, it induces great œdema of the glottis, necessitating tracheotomy, or it leads to necrosis of the cartilages and permanent disease of the larynx.[u] (See *Anatomical Lesions*).

8. *General Emphysema.* Chomel alluded to several instances where the body, soon after death, was found extensively emphysematous; and, in some, the emphysema had existed prior to death.[x] This complication was referred to by other writers, and was observed by the army-surgeons in the Crimea; but no satisfactory explanation was given of its origin. In 1857, Dr. Wilks exhibited a preparation to the Pathological Society, which accounted for the phenomenon in question. A boy, aged 12, became emphysematous on the twelfth day of an attack of 'typhoid fever,' the emphysema commencing in the neck, spreading to the face, arms, and chest, and greatly impeding deglutition. Death occurred on the twenty-second day, when it was found, that the air had escaped through a sloughing ulcer of the larynx, situated posteriorly, at the junction of the vocal cords.[y]

b. Diseases of the Organs of Circulation.

1. *Phlegmasia dolens* or *White Leg* is occasionally observed during

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[s] Trousseau, 1861, p. 200.
[t] Wilks, *Trans. Path. Soc.* ix. 34; xi. 14; Jenner, 1849 (2).
[u] See Trousseau, 1861, p. 197. [x] Chomel, 1834.
[y] *Trans. Path. Soc.* ix. 34.

convalescence, or even before the lenticular spots disappear, but is rarely the cause of much constitutional disturbance. In my experience, it has been a much more common sequela of pythogenic fever, than of typhus; but a contrary opinion has been expressed by Dr. Stewart.[z] The remarks on this subject, under the head of typhus, are equally applicable here, and make it very improbable that the affection is due, as has been suggested,[a] to phlebitis produced by absorption of pus from the intestinal ulcers. (See page 186.)

2. *Pyæmia.* Louis,[b] Forget,[c] and Peacock,[d] have recorded cases, where collections of pus have formed in different parts of the body, which have been attributed to phlebitis. I have met with several cases of this sort, and, in one instance, I have known the elbow-joint become filled with pus; but I know of no cases where pus has been deposited in the joints, with the formidable symptoms, described as occurring in typhus. (See page 189.)

c. Diseases of the Nervous System.

1. *Mental Imbecility and Mania.* After severe and protracted attacks of pythogenic fever, more or less fatuity occasionally remains during convalescence. The patient exhibits a childishness of manner and want of memory, and is the subject of various delusions. One little girl, under my care, believed the nurse to be her aunt, and some of the other patients, her sisters: while another patient was under the impression that he had inherited a fortune, with which he intended to enrich the hospital. Bartlett quotes the case of a young man, who had previously borne a good character; but who, after recovery from a grave attack of pythogenic fever, exhibited a strong propensity to steal.[e] This fatuity, which probably depends on an anæmic or atrophied condition of the brain, may last for many months; but I know of no case where it has been permanent. It is a more common sequela of pythogenic fever, than of typhus.

Occasionally, but much more rarely, the patient, during convalescence, is suddenly seized with violent delirium, independently of any cerebral inflammation. There is no heat of skin, or elevation of the pulse, and the delirium soon ceases.[f]

2. *Tubercular Meningitis.* Trousseau records a very rare case, where pythogenic fever was complicated with tubercular meningitis.[g]

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[z] Stewart, 1857. [a] J. R. Bennett, 1857. [b] Louis, 1841, case 15.
[c] Forget, 1841, cases 45 and 46. [d] *Med. Times & Gaz.* Ap. 26th, 1862.
[e] Bartlett, 1856, p. 51. [f] Trousseau, 1861, p. 189.
[g] *L'Union Méd.*, Aout, 6, 1859.

3. *General Convulsions* (see page 492).

4. *Chorea.* Barthez and Rilliet mention the case of a child, who was seized with chorea during convalescence and died: the spinal cord was found softened.[h]

5. *Paralysis,* either general or partial, may be observed after the cessation of the primary fever, in severe cases; but it is rarely permanent. Incontinence of urine is common in children. West mentions the case of a child, who had hemiplegia after convulsions, but who ultimately recovered.[i] M. Beau[k] has written a memoir on 'acute general paralysis' occurring as a sequel to enteric fever: the cases were invariably fatal, and I am inclined to think that the phenomena, which he described, were merely those of the typhoid state. (See also page 193.)[l]

d. Diseases of the Organs of Special Sense.

1. *Otorrhœa* is not an uncommon complication or sequela, particularly in children.[m] I have seen many cases. Suppuration of the internal ear occasionally terminates in meningitis. Cases of this nature are recorded by Louis[n] and Peacock.[o]

2. *Slight Amaurosis* occasionally occurs during convalescence.

e. Diseases of the Organs of Digestion.

1. *Pharyngitis.* Dysphagia, in severe cases, may result from muscular paralysis; but, in 8 out of 100 cases, I have noted that the patients complained of pain and difficulty in swallowing, at an early stage of the disease, when there was no great prostration. In most of these cases, the lining membrane of the fauces was very red. All recovered. Louis noticed dysphagia in 10 out of 46 fatal cases, and in 13 of 55 cases, which recovered. In the latter, the fauces were much injected; in the former, recent disease was found, after death, in the pharynx or œsophagus, such as ulceration, diphtheritic exudation, etc. M. Taupin says that dysphagia, in children, is often purely nervous, and independent of disease of the pharynx or œsophagus; attempts to swallow induce a spasmodic cough, resulting in the rejection of fluids by the nose.[p]

2. *Vomiting.* Constant vomiting after food, is occasionally observed during convalescence, particularly in patients who have been greatly reduced by the febrile attack. Trousseau regards this vomiting as of a nervous character, and says that it is best treated by giving solid food.[q]

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[h] Barthez and Rilliet, 1853, ii. 707.
[i] West, 1848, ed. 1854, p. 560.
[k] Beau, 1852.
[l] See also Trousseau, 1861, p. 191.
[m] Barthez and Rilliet, 1853.
[n] Louis, 1841, ii. 92.
[o] Peacock, 1856 (No. 1).
[p] Taupin, 1839.
[q] Trousseau, 1861, p. 188.

3. *Diarrhœa.* Occasionally, the intestinal ulcers, instead of cicatrizing, persist after the primary fever has ceased, and may give rise to exhausting diarrhœa, or proceed to perforation. In all cases, where there is obstinate diarrhœa after the fourth week, and after the disappearance of the eruption, the existence of these 'atonic ulcers,' as Rokitansky styles them, is to be apprehended.

4. *Dysentery.* Forget records a case of pythogenic fever, which proved fatal from dysentery, coming on during convalescence,[r] and Lyons has noted the occasional co-existence of the lesions of 'typhoid fever,' with those of dysentery.[s]

5. *Jaundice.* I have never met with jaundice in pythogenic fever, and Dr. Jenner makes a similar remark. Two fatal cases, however, are recorded by Louis;[t] two are reported by Frerichs;[u] and one case is mentioned by Jenner, which occurred on the West Coast of Africa.[x] All these cases proved fatal. In one case, reported by Frerichs, the jaundice appeared as early as the fifth day, and death occurred on the eighth day, before ulceration had commenced in the intestines. The pathology of the jaundice is the same as in typhus. (See page 196.)

6. *Ulceration of Gall-Bladder* (see *Anatomical Lesions*).

7. *Peritonitis* is a frequent complication of pythogenic fever, and may result from various causes.

a. The most common is perforation of the bowel, to be presently considered.

b. The inflammation may be propagated by continuity, from the mucous, to the peritoneal, coat of the bowel, without any perforation. In this way, peritonitis has been known to occur, as early as the ninth day, before ulceration had commenced. Most cases of peritonitis, which terminate favourably, are probably produced in this way.

c. Peritonitis may result from the bursting of softened morbid deposits in the spleen into the peritoneum. Two examples are recorded by Dr. W. Robertson;[y] and one, by Dr. Jenner.[z]

d. It may be produced by the bursting into the peritoneum of a softened mesenteric gland. Dr. Jenner records a case of this sort, in which recovery took place, under large doses of opium; but the patient died afterwards from erysipelas of the face.[a]

e. Lastly, fatal peritonitis may result from ulceration of the gall-bladder, ending in perforation. Barthez and Rilliet mention a case, where this happened in a girl, twelve years of age;[b] and

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[r] Forget, 1841, p. 351. [s] Lyons, 1861, p, 252. [t] Louis, 1841, obs. 17 & 26.
[u] *Dis. of Liver*, Syd. Soc. Transl. i. 172, 215. [x] Jenner, 1853, p. 312.
[y] W. Robertson, 1848. [z] Jenner, 1853, p. 287.
[a] Jenner, 1850, xxii. 405. [b] Barthez and Rilliet, 1853, ii. 5, 701.

a similar case occurred at the London Fever Hospital. The patient was a youth, aged 19, who was seized with symptoms of peritonitis, on the fifteenth day of the fever, and died within twenty-six hours. (See *Gall-Bladder* under *Anatomical Lesions*.)

It is impossible, during life, to distinguish between these different forms of peritonitis; but, in the great majority of cases, the cause is perforation of the bowel.

8. *Perforation of the Ileum*, with escape of the intestinal contents into the peritoneum, is the most important and dangerous complication of pythogenic fever. It occurs in the course of no other *acute* disease, the only other affection which gives rise to perforation in this part of the bowel being tubercular ulceration.

In one case at the Fever Hospital, the perforation took place as early as the ninth day of the fever. Peacock mentions a case, where it occurred on the eighth day;[c] and in one of Louis's cases, it happened on the twelfth day. In most cases, however, perforation does not occur until the third or fourth week, and in many it is delayed until after the cessation of the primary fever, being then produced by the so called atonic ulcers. Louis records a case where the date of perforation was the forty-second day; and Jenner another, where it was about the forty-sixth day; and, occasionally, it is even later. Of 32 cases observed by Louis (8),[d] and Bristowe (9),[e] and at the London Fever Hospital (15), the perforation occurred during the second week, in 8 cases;[f] during the third week, in 6; during the fourth week, in 9; and, after the fourth week, in 9.

The liability to perforation, after the cessation of the primary fever, is a point which cannot be too strongly insisted upon. Dr. Tweedie says, that he has known it occur when convalescence was supposed to be progressing so surely and satisfactorily, that the patient was allowed to leave the house, and when the stools were formed, and perfectly healthy in appearance.[g]

A large proportion of the cases in which perforation is met with, belong to that variety to be described as *latent* pythogenic fever, the bowels being scarcely, if at all, relaxed, and all the symptoms being of such a mild character, that the patient is able to go about almost to the date of the accident. In 10 out of 12 cases cited by Louis and Chomel, the disease was of this latent character, prior to the perforation.[h] Whether the case has before been latent or not, the advent of perforation is usually character-

[c] Peacock, 1856, (No. 1). [d] Louis, 1841, ii. 325. [e] Bristowe, 1860.
[f] In all, except one, it occurred on the 12th, 13th or 14th day.
[g] Tweedie, 1860, p. 159. [h] Chomel, 1834.

ized by the sudden supervention of collapse, and by acute pain in the abdomen, which, at the same time, is exquisitely tender, tense and tympanitic. Vomiting is one of the earliest symptoms, and is often preceded, accompanied, or followed, by urgent diarrhœa,[i] with or without intestinal hæmorrhage. The decubitus is dorsal, with the legs drawn up; the pulse is rapid, thready, or imperceptible; and the breathing is thoracic; the countenance is pale and pinched, and expressive of great suffering, and there is severe thirst. Soon the prostration becomes extreme; the extremities are cold; the face is covered with profuse sweats; and the patient gradually sinks, the mental faculties remaining unimpaired till the last. With such symptoms, the diagnosis of peritonitis can never be a matter of doubt.

But, occasionally, the symptoms are more obscure. Sometimes rapid sinking is the only change indicative of the supervention of perforation. Dr. Jenner reports a case, where the only symptoms were vomiting and coldness of the extremities, coming on eight hours before death. There was no pain, although the patient was sensible to the last.[k] The advent of perforation may likewise be latent, owing to the patient being delirious or unconscious, as happened in one of my cases, where death was preceded for two days by copious hæmorrhage from the bowels, but in which there was no sudden aggravation of the symptoms, no increased rapidity of the pulse, and no vomiting. In 3 out of 8 cases, recorded by Louis, the symptoms of perforation were obscure.

The occurrence of perforation is sometimes followed by death within three or four hours, and life is rarely prolonged beyond forty-eight hours. In one case, however, I have known the patient survive the occurrence of perforation 105 hours; in a second case, twelve days; and in a third case, sixteen days. Louis has recorded a case, where there was an interval of seven days; and Bristowe another, in which there was an interval of upwards of a fortnight, between the first symptoms of perforation and the fatal result.

Most observers, including Louis, Chomel, Rokitansky, and Jenner,[l] have expressed the opinion that perforation is invariably fatal, and, certainly, the cases are few where it is not so. At the same time, it is satisfactory to know, that rare cases are occasionally met with, where recovery ensues after all the symptoms of peritonitis from perforation. Dr. Tweedie states that he has wit-

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[i] Diarrhœa does not always cease suddenly, on the occurrence of perforation, as has been stated. [k] JENNER, 1850, xxii. 298. [l] Ibid. 1853, p. 286.

nessed the recovery of two cases, in which the distinctive signs of perforation were unequivocal, and the late Dr. Todd informed me that he had observed a similar case. Another case of recovery is mentioned by Dr. E. L. Fox,[l] another by Mr. Ballard,[m] and several are reported by Dr. J. Bell, of Glasgow, as having occurred in his practice.[n] One case has come under my own observation. The patient was a girl 15 years of age, who, on the thirty-first day of the disease, was suddenly seized with severe pain and tension of the abdomen, urgent vomiting, and all the symptoms of collapse. A grain of opium was ordered every second hour, and during the first thirty-six hours ten grains were taken. The patient made a tedious recovery, and was discharged from the hospital fifty-five days after the commencement of the peritonitis.

It may be doubted if the peritonitis, in these cases of recovery, has been due to perforation of the bowel. Trousseau[o] and Jenner record cases where it was found, after death, to be independent of perforation, although all the symptoms of peritonitis from perforation had existed during life. Taking a pathological view of the question, there appears to be no reason why recovery should not occasionally take place. I have repeatedly found a minute perforation, with its edges glued to the neighbouring parts, in such a way, that little or no escape of the intestinal contents had taken place, and where, in fact, a natural process of cure appeared to be commencing. When the perforation is very large, the case is probably, invariably fatal. But even when the opening is large enough to allow some of the intestinal contents to escape, the peritonitis may be limited by adhesions, so that a *circumscribed peritoneal abscess* results, and then a long period may intervene between the occurrence of perforation and the fatal event, and recovery is, perhaps, not impossible. Dr. Bristowe has recorded the case of a girl, who recovered after paracentesis, from an attack of circumscribed suppurative peritonitis, which was attributed to perforation of the bowel in enteric fever.[p] In another case, I have known a circumscribed peritoneal abscess, forming a perceptible tumour in the abdomen, follow an attack of peritonitis in enteric fever; pus was passed for a long time from the bowel, and the patient was confined to bed for nine months, but eventually recovered. (See *Anatomical Lesions.*)

Intestinal perforation, in enteric fever, is probably more common than is generally believed. Of 165 autopsies of enteric fever,

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[l] *Brit. Med. Journ.* June 8th, 1861.
[m] *Lancet*, 1860, i. 422. [n] Bell, 1860, viii. 388. [o] Trousseau, 1861, p. 142.
[p] Bristowe, 1860, p. 115.

recorded by Waters,[q] Jenner, Bristowe, or made at the London Fever Hospital, I find that perforation occurred in 35, or in 21·2 per cent. Again, of 270 autopsies recorded by Bretonneau,[r] Louis, Chomel, Montault,[s] and Forget,[t] perforation existed in 25, or 9·25 per cent. Adding the French and English results together, we have 60 cases of perforation in 435 autopsies, or perforation occurred in 13·8 per cent. Taking the mortality from enteric fever, in England and France, at about 20 per cent., it follows, that 1 in every 36 persons attacked with enteric fever dies of perforation. The English results alone would make the occurrence more frequent.[u] The above conclusion coincides with what has been observed on a small scale, in actual practice. Perforation occurred in 2·74 per cent. of Dr. Flint's cases,[x] and in 3 of my 100 cases, of which the symptoms are analysed in this work.

Perforation appears to be more common in males than in females. Of 15 cases observed by Bristowe, 11 were males. Of 24 cases at the London Fever Hospital, 16 were males, although of the total cases fatal from enteric fever the number of females was slightly in excess of that of males.

The age at which perforation occurs was noted by Louis, Chomel, Bristowe, and at the London Fever Hospital, in 44 cases, as follows:—Under 10 years, in 1; from 10 to 15, in 1; from 15 to 20, in 18; from 20 to 25, in 7; from 25 to 30, in 10; from 30 to 40, in 6; and 45, in 1. Messrs. Barthez, Rilliet, and Taupin[y] met with perforation in 3 out of 232 children under treatment.

Case XXXV.

Pythogenic Fever. Acute Delirium and Unconsciousness. Profuse Intestinal Hæmorrhage, and Death on the nineteenth Day. No Symptom of Peritonitis. Autopsy:—Ulceration of Intestines; Perforation; Peritonitis.

James L——, aged 19, a mechanic in good circumstances, was admitted into the London Fever Hospital on August 19th, 1858, having been ill for about eight days. His bowels had been much relaxed, and for two days before admission, he had been very delirious.

August 20th (tenth day). Pulse 120, full, but compressible. Says he has slight headache, and he is rather confused. Was very delirious in the

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[q] Waters, 1847. [r] Bretonneau, 1829. [s] Montault, 1838. [t] Forget, 1841.

[u] During ten years (1840-49), Heschl found perforation in only 56 of 1271 autopsies, or in less than 5 per cent. It may be doubted, however, if he did not include cases of typhus, for the same observer in 1852-53 found perforation in no fewer than 11 of 72 autopsies of 'typhus' (see Heschl, 1853).

[x] Bartlett, 1856, p. 60. [y] Barthez and Rilliet, 1853, ii. 701.

night, and attempted to leave his bed several times. Several lenticular rose-spots on chest and abdomen. Tongue moist and furred, and red at edges; intense thirst; great tympanitis and tenderness in right iliac fossa; two light watery stools. Was ordered beef-tea and milk, and mistura camphoræ.

August 21st (eleventh day). Pulse 132. Is more prostrate, and was again very restless and delirious in the night. Skin hot and dry; temperature in axilla 104° Fahr. Lenticular spots more numerous; tongue dry along the centre, and red at edges; abdominal tenderness increased; five watery motions. Was ordered a turpentine stupe to the abdomen; acetate of lead (gr. iij.) every four hours; a starch and laudanum enema at night, and 4 ounces of brandy.

August 24th (fourteenth day). Pulse 144, and weak. Is now unable to get out of bed, but still tries to do so, when he is delirious at night. Is confused, but understands what is said to him; pupils natural; circumscribed flush on both cheeks; numerous lenticular rose-spots; fresh ones appear daily, while several marked on admission are no longer visible. Tongue red and moist; great tympanitis; two watery stools. Since August 22nd, the patient has been taking a mixture containing ammonia and chloric ether, instead of the lead, and he has had a morphia draught at night. To-day, the brandy was increased to 8 ounces.

August 26th (sixteenth day). Pulse 136. Scarcely knows his friends; moans and sighs very much; but always calls for bed-pan, when he requires it. Spots continue; skin is moist, and has perspired every night since admission, after which he has been very faint. Two stools.

August 27th (seventeenth day). Had no motion since yesterday, till this afternoon, when he passed a large quantity of fetid, liquid, red blood. No vomiting, and the tenderness of abdomen seems less than before; but patient is scarcely conscious. Was ordered a starch enema with 20 drops of laudanum, and a draught with 15 minims of turpentine every three hours.

August 28th (eighteenth day). No motion for some hours after the enema; but since then he has had five, consisting of nothing but pure blood. Tongue dry and brown; sordes on teeth; slight tenderness of abdomen. Pulse 136, small and weak; very noisy in the night, and scarcely knows his father; but got up to stool himself, when the nurse was not present.

August 29th (nineteenth day). Died at 7½ a.m. Was very restless and delirious until half an hour before his death. Passed one bloody motion in bed, in the night.

Autopsy, thirty-five hours after death.

Heart 10 ounces; permanent foramen ovale; small white coagulum in right ventricle. Slight hypostatic congestion of both lungs; right, 20 ounces; left, 17 ounces.

The abdominal cavity contained about half a pint of dirty yellow fæcal fluid. The peritoneal surface of the small intestines was very vascular, and coated with loosely adherent flakes of lymph. Twelve inches above

the ileo-colic valve was a semilunar perforation, measuring nearly 2 lines in its long diameter, and formed in this way:—An oval patch of peritoneum, measuring $4\frac{1}{2}$ lines by 2 lines, had sloughed, its smooth pale yellow surface contrasting strongly with the surrounding bright red membrane roughened by the deposit of lymph. This slough still adhered by its edges, except at one extremity, where it was detached, forming the semilunar perforation (see fig. 5). The little opening was plugged up by a

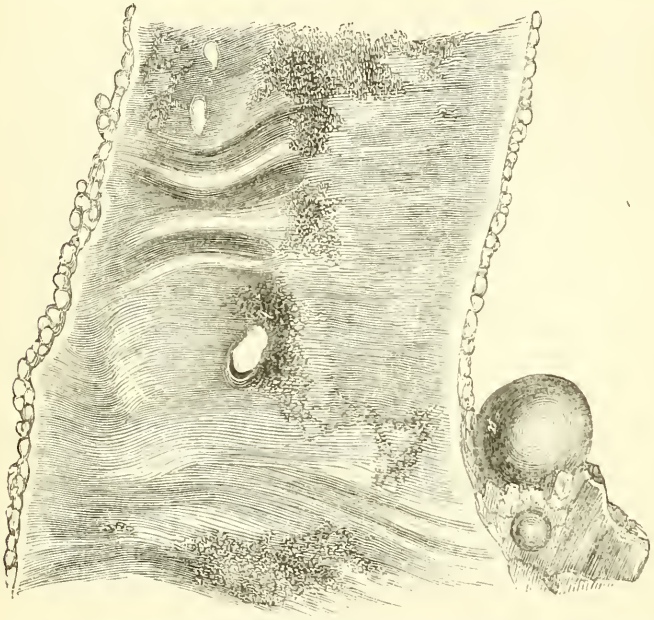

Fig. 5. Perforation of ileum, seen from peritoneal surface. *a*. Enlarged mesenteric gland *b*. Dead portion of peritoneum, surrounded by increased vascularity: at its lower end is the perforation. *c*. Flakes of lymph.

fragment of slough from the interior of the bowel. On slitting open the intestine, the lower four inches of the ileum were found to be one mass of ulceration, which terminated abruptly at the valve. This ulcerated surface was covered with loosely attached yellowish sloughs, and with masses of coagulated blood. Six of Peyer's patches, and many of the solitary glands above this, were ulcerated, yellowish sloughs being still loosely attached to most of the ulcers. In one of Peyer's patches, was the perforation already described. Some of the solitary glands were enlarged from morbid deposit, up to the size of a split-pea, but were not ulcerated. Many of the solitary glands in the cæcum and ascending colon were either ulcerated, or contained morbid deposit. The large intestine contained a few ounces of blood. The mesenteric glands were much enlarged, some of those near the cæcum being as large as a pigeon's egg. Liver 60 ounces, anæmic, but healthy; 12 drachms of very pale, watery

bile in gall-bladder. Spleen, 9 ounces, dark, and rather firm. Kidneys large, and very congested; right, 6½ ounces; left, 5 ounces.

Case XXXVI.

Pythogenic Fever of moderate Severity. After temporary Improvement, Peritonitis and Stercoraceous Vomiting on 23rd day, and Death within 36 hours. Autopsy:—Ulceration of Intestines. Perforation and Peritonitis.

Thomas P., aged 21, was admitted into the Fever Hospital, Sept. 15th, 1858. He began to complain of giddiness, headache, and pains in the limbs, on Sept. 1st, and almost from the first, his bowels had been much relaxed.

Sept. 15th, pulse 108. No headache. Intelligence clear; pupils dilated. Several lenticular rose-spots on chest and abdomen. Temperature under tongue 104° Fahr. Tongue red; abdomen tympanitic; bowels still relaxed. Was ordered beef-tea and milk, and acetate of lead (gr. iij.), after each motion of bowels.

Sept. 16th (16th day). Pulse 92. Is more prostrate, but can get up without assistance. Is restless at night, and mutters in sleep; but intelligence is clear, when awake. Tongue dry and brown, along the centre; 4 light, watery stools. Ordered 6 ounces of wine.

For the next four days, the patient continued much in the same state, except that, on Sept. 18th, he became a little drowsy. His intelligence always seemed clear when he was spoken to; the pupils were always dilated; fresh spots continued to appear daily in large numbers; the bowels were moved two or three times daily, and the pulse never exceeded 96.

September 20th. Pulse 120, but patient says he feels much better. Three motions.

September 21st (twenty-first day). Pulse 88. Intelligence clear, but is more drowsy. Temperature under tongue only 102° Fahr.; 105 rose-spots counted on front of chest and abdomen. Lips parched. Tongue dry, red, smooth, and deeply fissured. Less tympanitis, and scarcely any tenderness of abdomen; three stools. Appearance much improved.

September 23rd (twenty-third day). Pulse 120, and weaker, and does not feel quite so well. Slept well, but is a little confused; pupils dilated; 21 fresh spots on front of chest and abdomen, and many of old ones gone. Only two stools, which are of more consistence. Ordered 6 ounces of brandy.

At 6 p.m., urgent diarrhœa came on; and in the night, he was seized with acute pain in the abdomen, followed by vomiting.

September 24th. Pulse 108, very weak; features pinched; very prostrate, but is perfectly conscious. One stool since midnight, passed in bed. The vomited matters exactly resemble the fæces, both in smell and colour; abdomen tense, and very tender; spots numerous. A starch and laudanum enema was administered, and a grain of opium was ordered

every second hour. The diarrhœa and vomiting continued, and at 8 a.m., of the 25th, the patient died, his mind remaining clear to the last.

Autopsy, 8 hours after Death.

Cadaveric rigidity marked. None of the lenticular spots visible, although their situation is indicated by circles of ink.

A small mass of pale lymph in right cavities of heart. Both lungs healthy; right, 16 oz.; left, 16½ oz.

The abdomen contained about a pint and a half of opaque yellowish fluid, containing flakes of lymph, but apparently not fæcal. The whole of the intestines were glued together by recent lymph, which also coated the surface of the liver and the under-surface of the diaphragm. The peritoneal surface of the intestines was intensely injected, especially over the lower six feet of the ileum. The portions corresponding to Peyer's patches were particularly bright, and here also the lymph was more adherent than elsewhere. On slitting up the intestine, an ulcer was found corresponding to almost every one of Peyer's patches, in the lower four feet of the ileum. The sloughs had separated from all of them, and in several, the floor was formed by the transverse muscular fibres, or by the peritoneum. In one of the ulcers, 5½ inches above the valve, was a small circular perforation, barely large enough to admit a stocking wire. The peritoneal edges of this perforation were glued by lymph to a neighbouring coil of bowel, so that the contents of the bowel had been prevented from escaping in any great quantity. None of the solitary glands in the large intestine, and but few in the ileum, showed any trace of disease. Mesenteric glands enlarged; but none larger than a hazel nut. Spleen 14 ounces, dark and rather soft. Liver, 73 ounces, fatty; one ounce of pea-green bile in gall-bladder. Kidneys enlarged; each 6 ounces; hyperæmic.

f. Diseases of the Urinary Organs.

1. *Disease of Kidneys.* As in typhus, disease of the kidneys, of such a nature as to impair their functions, is always a very serious complication (see page 197).

g. Complications referrible to the Organs of Generation.

1. *Pregnancy.* According to Rokitansky,[z] pregnancy offers an almost entire immunity from pythogenic fever; but the correctness of this opinion has been denied by Jenner,[a] Forget, etc. I have met with several instances of pregnant females who were attacked with the disease. If the pregnancy be advanced, the case is almost certain to be fatal, death being usually preceded by abortion. In one of my cases, the patient survived the primary attack, which was severe, but died during a relapse (see pages 197 and 361).

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[z] *Path. Anat.* Syd. Soc. Transl. ii. 82.

[a] JENNER, 1850, xxii. 439.

h. Diseases of the Integuments, etc.

1. *Erysipelas* is even more common than in typhus. Louis noted it in 9 of 134 cases; Chomel in 4 of 42 fatal cases; and Jenner in 7 out of 23 fatal cases. It usually appears in an advanced stage of the disease, or in convalescence, and is often fatal; 10 of the 13 cases observed by Louis and Chomel, died (see page 197).

2. *Anasarca.* Slight œdema of the lower extremities is occasionally observed, during convalescence from protracted attacks. It results from debility, and is not associated with albuminuria.

Professor Leudet has published an account of seven remarkable instances of 'typhoid fever,' observed at Rouen, where the inferior extremities, and the entire body, became very œdematous in the second or third week of the fever, or during convalescence. All the patients but one, who died of peritonitis, recovered; none had albuminous urine. The swelling was unattended by pain, but was ushered in by severe bronchitis and profuse sweating. After lasting for two or three weeks, it disappeared, and it gave rise to no inconvenience, except that it retarded convalescence. The cause of the dropsy in these cases was believed to be the adynamic constitution of the persons attacked.[b] Similar cases were observed at Tübingen, by Griesinger, in persons who had been very destitute prior to their attack of fever.[c] Barthez and Rilliet also speak of general or partial anasarca as a not uncommon sequela of pythogenic fever in children. It occurred in 7 out of 111 cases, which they analysed. In 2 other of their cases, extreme general anasarca came on as early as the fifth day of the fever, and lasted eight or ten days. There was no albumen in the urine; after the disappearance of the œdema, the fever ran its usual course. Both children recovered.[d]

3. *Gangrene from Pressure.* Bed-sores are much more common in pythogenic fever, than in typhus. This circumstance has been dwelt on by most observers, who have had much experience in both diseases,[e] and is readily accounted for by the greater emaciation in the former malady, and its longer duration. In children, bed-sores are comparatively rare; but they were observed in 6 out of 107 cases, by Barthez and Rilliet (see page 198).

4. *Spontaneous Gangrene,* on the other hand, is less common than in typhus, but is occasionally met with. Three cases of gangrene of the lower extremities are mentioned by Trousseau, who ascribes it to obliteration of the arterial trunks; in none,

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[b] Leudet, 1858. [c] Griesinger, 1857.
[d] Barthez and Rilliet, 1853, ii. 707. See also Trousseau, 1861, p. 192.
[e] Barrallier, 1861, p. 96.

was there an opportunity of examining the artery after death; but in one of the cases, the posterior tibial artery could be felt as a hard, painful, pulseless cord.[f] Trousseau also records cases, in which sloughing of the corneæ occurred, in consequence of constant exposure of the eyes, from paralysis of the orbiculares muscles.[g] Sloughing of the penis has been known to occur, in cases where the patient has been suffering from syphilis or gonorrhœa before the febrile attack (see page 198).

5. *Noma*, or *Cancrum Oris*, is not common in pythogenic fever, but it is occasionally met with in children. Two cases are mentioned by West;[h] one has come under my own observation; and of 98 cases of gangrene of the mouth, observed by M. Tourdes, 7 followed on 'typhoid fever.'[i] It is usually fatal (see page 199).

6. *Ulceration from Blisters.* Louis pointed out, that blisters, in pythogenic fever, were very slow in healing, and were apt to degenerate into unhealthy sores;[k] and his experience has been confirmed by that of all subsequent observers.

7. *Inflammatory Swellings or Buboes.* Collections of pus, in different parts of the body, are occasionally met with after severe attacks of pythogenic fever; but the hard inflammatory swellings, in the region of the parotid, and elsewhere are comparatively rare. In only 1 case have I met with a characteristic parotid bubo, such as I have so often observed in typhus (see page 200). Louis[l] and Chomel[m] each record 1 case, and another is reported by Dr. W. T. Gairdner.[n] Chomel regarded these swellings as critical and auspicious; but Trousseau observes that he has scarcely ever known a case recover, in which they appeared.[o]

i. Marasmus.

In certain cases of pythogenic fever, the vital powers remain in a depressed state after the attack; the patient does not gain flesh and has a repugnance to food, or he may eat well, but the food does not appear to be assimilated; and yet no local disease can be discovered. I have known a case prove fatal, in this way, eight weeks after the cessation of the primary fever, where no local lesion could be found after death. Rokitansky alludes to this depression of the vegetative system, passing into genuine *tabes*, and says that such cases are characterized by a shrivelled

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[f] Trousseau, 1861, p. 211. [g] Ibid. p. 194.
[h] West, 1848, ed. 1854, p. 561. [i] Barthez and Rilliet, 1853, ii. 704.
[k] Louis, 1841, ii. 124, 483. [l] Ibid. II, 97, 371.
[m] Chomel, 1834. [n] Gairdner, 1862 (2), 141. [o] Trousseau, 1861, p. 170.

condition of the mesenteric glands, and by a loss of the villi and follicles of the intestine.[p]

k. Other Specific Diseases.[q]

1. *Pythogenic Fever and Scarlatina.* Patients admitted into the London Fever Hospital, with pythogenic fever, have not unfrequently contracted scarlet fever from other patients in the same ward; and I have notes of 8 cases, in which the eruptions of the two diseases co-existed. The following are the histories of two such cases, and others will be found in my memoir, on the Co-existence of Specific Morbid Poisons.[r] Similar cases have also been recorded by Taupin[s] and Forget.[t]

Case XXXVII.

Co-existence of Scarlatina and Pythogenic Fever.

A policeman, aged 23, was admitted into the London Fever Hospital November 9th, 1857, having been ill for two or three weeks. On admission, he had all the symptoms of pythogenic fever, including a red, glazed, and fissured tongue, tympanitis, profuse watery diarrhœa, and very numerous lenticular spots. Fresh spots continued to appear; and, eight days after admission, they were still very numerous, and the diarrhœa persisted. There was now, in addition, a general scarlet rash, identical with that of scarlet fever, a strawberry-red tongue with large papillæ, sore throat, and redness of the fauces. Two days later, the lenticular spots were still very numerous, and the scarlet rash persisted. Two days after this, the scarlet rash was fading, but the lenticular spots continued out for a few days longer. A week after the disappearance of the scarlet rash, there was copious desquamation. The patient made a good recovery.

Case XXXVIII.

Co-existence of Scarlatina and Pythogenic Fever.

A boy, aged 14, was admitted into the London Fever Hospital, August 25th, 1858, from a house, in which there had been other cases of pythogenic fever. He had all the ordinary symptoms, in a mild form. Lenticular spots appeared on the thirteenth day of the fever, and continued coming out in successive crops. On the twenty-second day, there were still several spots, and also a general bright scarlet rash, having all the characters of that of scarlet fever. The tongue, which before had been almost clean, became covered with a thick white fur, through which could be seen large red papillæ; the throat was sore; and the tonsils were enlarged and red, and coated with a white membranous exudation. On the

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[p] *Path. Anat.* Syd. Soc. Transl. ii. 81. See also, Huss, 1855, p. 221.

[q] See *antea* page 209. [r] Murchison, 1859, No. 4, p. 194. [s] Taupin, 1839, p. 245.

[t] Forget, 1841, p. 146. See also *Gaz. Méd. de Paris*, 1833, p. 765.

same day, the pulse was found to have risen from 72 to 132, and the temperature under the tongue from 99° to 104° Fahr. Both the eruptions continued distinct for four days, and then disappeared. On the twenty-fifth day, the tonsils were so large, as almost to meet, and the tongue was clean, red, and of a strawberry aspect. On the twenty-seventh day, desquamation commenced. Convalescence was delayed by glandular swellings in the neck, one of which terminated in abscess. After this boy's admission, a patient with scarlet fever lay in the adjoining bed, and there were many other cases in the same ward.

2. *Diphtheria.* Louis,[u] Forget,[w] Barthez and Rilliet[x] record cases, showing the co-existence of pythogenic fever and diphtheria, and one case, of the same nature, has occurred in my own practice.

3. *Rubeola.* Barthez and Rilliet, and M. Taupin mention cases, in which pythogenic fever and rubeola coexisted.[y]

4. *Typhus* (see Chapter V).

Section IX.—Varieties of Pythogenic Fever.

There is no disease which presents itself under a greater variety of forms, than pythogenic fever. These varieties are due to, 1, differences in the severity of the disease, as a whole; 2, the absence or unusual development of certain symptoms; and 3, the presence of complications. They are accounted for by differences in the age or constitution of the patient, or in the intensity of the poison. I have often been struck with the similarity in the symptoms, of all the cases occurring in the same house. Thus, I have known all the cases in one house very mild, and in another, very severe; urgent diarrhœa or sickness in one house, no diarrhœa or sickness in another; severe cerebral symptoms in one house, no cerebral symptoms in another; and in one instance, I have met with three cases of relapse in the same family. The following are some of the principal varieties of pythogenic fever, met with in practice. Some of them have been regarded as distinct diseases.

1. *The Mild Form* is the most simple. The characters, by which it is distinguished from the other forms, are chiefly negative. The main symptoms are slight fever, lenticular rose-spots, diarrhœa, and disturbed sleep. The appetite may be but slightly impaired; and, from first to last, there may be no delirium or somnolence. This corresponds to the *forme muqueuse* of French writers. Even

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[u] Louis, 1841, i. 135, 513.
[w] Forget, 1841, p. 339.
[x] Barthez and Rilliet, 1853, ii. 706.
[y] Taupin, 1839, p. 245.

diarrhœa may be absent; and the symptoms altogether may be so trifling, that the real nature of the affection is not recognized, or it is mistaken for remittent fever, or, if its duration be short, for Febricula.

2. *The Grave Form* of the disease comprises many sub-divisions. First, there is the so-called *inflammatory form*, when the skin is very hot, the pulse quick and of good strength, the thirst intense, and the urine scanty and dark. Secondly, there is the *ataxic form*, characterized by acute delirium, followed by the typhoid state (see page 497). This variety, like typhus, is vulgarly designated *Brain-fever*. Thirdly, the *adynamic or low nervous form*, is distinguished by great prostration, feeble pulse, low muttering delirium, and involuntary evacuations. Fourthly, there is the *irritative form*, where the patient is restless, sleepless, anxious, and intolerant of light and sound. Fifthly, there is the *abdominal form*, in which the abdominal symptoms are most prominent; and sixthly, there is the *thoracic form*, in which the symptoms are modified by thoracic complications. Lastly, there is the *hæmorrhagic* form, or the *Fièvre putride hémorrhagique*[z] of Trousseau and other French writers, in which there is a remarkable tendency to hæmorrhages from the various mucous surfaces, and extravasations beneath the skin, in the form of petechiæ and vibices.

These distinctions apply only to typical cases. Numerous combinations and gradations of the different varieties of the grave form of the disease are constantly met with.

3. *The Insidious,* or *Latent Form,* is a most important variety of pythogenic fever. It was well described by Dr. Hewett of London, in 1826;[a] and it has been prominently noticed by Louis, Chomel, and many other writers. In this form, all the symptoms are at first of a very mild nature. There may be no great acceleration of the pulse, and the characters may be so indefinite, that the nature of the case is not diagnosed. In one class of cases, the chief symptoms are irregular chills, alternating with heat and flushing, slight headache, loss of appetite, lassitude, and disturbed sleep: diarrhœa may be absent, or the bowels may be constipated. In another class of cases, the patient suffers chiefly from catarrh, and he is thought to have merely 'taken a cold.' In a third class, the chief symptoms are nausea, vomiting, and a red tongue, and the patient is thought to be suffering from gastric derangement or from slight 'gastric fever.' The above symptoms continue for two weeks, or longer, but are so mild, that no anxiety is felt about the

[z] Trousseau, 1861, p. 152. [a] Hewett, 1826.

patient, when suddenly, he becomes alarmingly ill; profuse hæmorrhage from the bowels takes place, which may induce fatal syncope; or, more commonly, symptoms of peritonitis from perforation set in, and, after a few hours, terminate in death. Cases, answering to this description, are not uncommon. Before the alarming symptoms occur, the patient's prostration may be so slight, that he is able to follow his ordinary avocations until within a few hours of the fatal event. I have known a man walk more than a mile to the London Fever Hospital, at the end of the third week of the fever, and die from perforation, in less than thirty hours after his admission. Louis mentions the case of a man, who walked daily in the hospital-garden, up to the twenty-third day, when perforation occurred, which was followed by death in thirty-six hours.[b] In most such cases, indeed, the perforation is probably due to rupture of the denuded peritoneum, forming the base of the intestinal ulcers, which would not have happened, if the primary symptoms had been sufficiently severe, to compel the patient to maintain the recumbent posture.

4. *Infantile Remittent Fever.* It has long been known, that children are very liable to fever, accompanied by gastric and intestinal disorder, and the designations, Worm Fever, Infantile Hectic, Infantile Gastric, and Infantile Remittent Fever, have been applied to it. Abercrombie,[c] Wendt,[d] Billard,[e] Meissner,[f] Evanson and Maunsell,[g] accurately described both its symptoms and anatomical lesions; but they regarded the fever, as symptomatic of the local disease, and as peculiar to children. So little was it thought that the disease was the same as the enteric fever of adults, that Chomel, in 1834, wrote concerning the latter affection, as follows: 'Nous ne craignons pas de nous tromper en 'disant, que ce nombre va continuellement en diminuant jusqu'à 'l'âge de dix ans, au dessous duquel il paraît, que les enfans ne 'sont que très rarement atteints de cette affection.'

In 1836, M. Hutin[h] published the account of an epidemic of enteric fever in children; but it is to Messrs. Rilliet,[i] Taupin,[k] Löschner,[l] and Stœber,[m] and to the writings of our countryman, Dr. West,[n] that we are indebted, for establishing the identity of infantile remittent fever with the enteric fever of adults. It is now known, that children are particularly liable to enteric fever, as they are often attacked when other members of the same family

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[b] Louis, 1841, ii. 223. [c] Abercrombie, 1822. [d] Wendt, 1822.
[e] Billard, 1828. [f] Meissner, 1838. [g] Evanson and Maunsell, 1836.
[h] Hutin, 1836. [i] Rilliet, 1840. [k] Taupin, 1839. [l] Löschner, 1846.
[m] Stœber, 1841. [n] West, 1848.

escape. The symptoms and complications of the fever are modified, to some extent, by the age of the patient, as has already been shown; and in children, the remittent character of the disease is more marked than in adults.

It is a subject for enquiry, if all idiopathic remittent fevers, in children, are really examples of enteric fever. Dr. Handfield Jones is of opinion that certain cases, in which children are feverish, or even delirious at night, but free from fever in the daytime, are of a malarious character, and curable by quinine.[o] The question, however, can only be settled by *post-mortem* examinations, which are still wanting; but it may be mentioned that true enteric fever often assumes a remittent character, and is then sometimes benefited by quinine (see under *Treatment*).

5. *Gastric* or *Bilious Fever.* 'What is Gastric Fever?' is a question, which has been often put of late years, and to which many practitioners hesitate to reply. If we turn to the published descriptions of 'gastric fever,' there is no doubt about the matter. The gastric and stomachic fevers, described during the last century, by Ballonius, Heister, and Burserius,[p] were unquestionably enteric fever. Cheyne,[q] and Craigie,[r] were the first to employ the term 'gastric fever,' in this country, and their descriptions of the symptoms and anatomical lesions show plainly that the disease, which they had in view, was also enteric fever. Medical literature, in fact, contains nothing, on which to base the opinion, that there is an idiopathic gastric fever. Notwithstanding this, it is commonly believed that there is such a disease; and, in proof, cases are appealed to, where febrile symptoms have lasted for three or four weeks, or longer, and have been associated with gastric disturbance and vomiting, but not necessarily with diarrhœa. I have repeatedly met with cases answering to this description; but I have rarely failed to discover the characteristic spots of enteric fever, while the opinion, that they were merely mild cases of enteric fever, has been confirmed by other considerations. I have known cases of this nature occur in the same house, and at the same time, as undoubted examples of enteric fever. I have also met with cases which, for the first fortnight, would have been regarded by many as examples of gastric fever, but which ultimately passed into the typhoid state, and proved fatal, the lesions of enteric fever being discovered in the dead body. Such cases, indeed, are far from rare, as is shown by an expression in common use: 'gastric fever pass-

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[o] Jones, 1858 (2), and *Brit. Med. Journ.* Jan. 25th, 1862.
[p] Burserius, 1785. [q] Cheyne, 1833. [r] Craigie, 1837 (1).

ing into typhoid.' This expression itself is opposed to the individuality of gastric fever, for no person believes that one specific disease is convertible into another. Again, I have known a case run the course of so-called gastric fever in the primary attack (the bowels being confined, and the symptoms being mainly referrible to the stomach), and of well-marked enteric fever, in the relapse. For six years, I have earnestly looked for a case of gastric fever, but I have not succeeded in meeting with one. My opinion is, that in all cases of 'gastric fever,' the disease is really enteric fever, or that the febrile symptoms are due to derangement of the stomach and liver, from non-specific causes.

Since the above was written, I find that Dr. Anderson of Glasgow, in his valuable lectures, has described gastric fever as a distinct affection. Among its characters are mentioned: a fissured red tongue, a variable pulse, vomiting, abdominal pain, tympanitis, diarrhœa, and intestinal hæmorrhage; an eruption, consisting of 'a few small insignificant-looking red spots,' appearing about the seventh day; and after death, ulceration of the intestines. Dr. Anderson draws distinctions between the eruption, the stools, and the intestinal lesion of this fever, and those of enteric fever; but I must confess my inability to find, in his description, any characters on which to found a specific difference; and this conclusion is confirmed by the circumstance, that Dr. Anderson believes that his gastric fever is often traceable to the 'foul emanations from 'drains or cess-pools;' and that he claims, as belonging to it, the infantile remittent fever, which all modern pathologists regard as identical with enteric fever.[5]

Section X. Diagnosis of Pythogenic Fever.

During the first week of the disease, it is often difficult to form a positive diagnosis; but even then, pythogenic fever is to be suspected, if diarrhœa co-exists with frontal headache, disturbed sleep, and with general febrile symptoms, increasing towards night.

When, after febrile symptoms of about a week's duration, lenticular spots appear in successive crops, as described at page 468, the diagnosis of pythogenic fever is certain, whatever be the other symptoms. It may sometimes be a question, how many spots are necessary for a diagnosis. I believe that two or three are sufficient, provided it is ascertained by encircling them with ink, that they disappear after two or three days, while one or two fresh ones are developed elsewhere.

[5] Anderson, 1861, p. 122.

But if there be no spots, or if they be indistinct, the diagnosis of pythogenic fever is tolerably certain, in a case where febrile symptoms have lasted upwards of a week and are associated with diarrhœa, ochre-yellow stools, tympanitis and abdominal tenderness, enlarged spleen, and considerable muscular prostration. If, along with these symptoms, there be epistaxis, hæmorrhage from the bowels, or delirium, there can be no mistake as to the nature of the case.

If both the eruption and the abdominal symptoms be absent, the diagnosis of pythogenic fever can only be arrived at by a process of exclusion, or by carefully weighing the symptoms and circumstances of the individual case, and comparing them with those of the other diseases, with which it is most apt to be confounded. Under the circumstances mentioned, a diagnosis from many other diseases may sometimes be impossible.

The diseases, which are most apt to be confounded with pythogenic fever, are typhus, relapsing fever, remittent fever, variola, scarlatina, pyæmia, latent pneumonia, acute tubercle of the lungs, tubercular meningitis, cerebral softening, gastro-enteritis, and some forms of chronic peritonitis.

1. *Typhus.* The eruptions are the grand distinguishing marks between typhus and pythogenic fever. When they are present and well-developed, there can be no difficulty whatever in forming a diagnosis. When they are absent, it may be impossible to say to which fever a case belongs, or indeed, whether it be an example of Continued Fever at all. For example, if the typhoid state be developed when the patient first comes under observation, and if nothing be known as to the previous history, it may be impossible to decide whether the case is typhus, pythogenic fever, cerebral disease, or disease of the kidneys. It must also be remembered, that the mere existence of diarrhœa does not distinguish pythogenic fever from typhus. Typhus may be complicated with diarrhœa, and the bowels may be constipated in pythogenic fever. If, however, diarrhœa co-exist with tympanitis and pain in the right iliac fossa, and if the stools be ochrey-yellow, it may be concluded that the case is pythogenic fever; and this opinion will be strengthened by the occurrence of epistaxis, or of intestinal hæmorrhage. Again, the emaciated countenance and the circumscribed pink flush on either cheek, contrast strongly with the heavy expression, the dusky countenance, and the injected conjunctivæ of typhus. The diagnosis is also assisted by the appearances presented by the tongue (see pages 140 and 478), and pupil (see pages 169

and 493), by the duration of the illness (see pages 177 and 498), and by the circumstances under which the disease appears (see pages 71, 74, 423, 426). See also Chapter V.

2. *Relapsing Fever.* Cases of pythogenic fever, followed by a relapse, are occasionally designated *Relapsing Fever.* The clinical histories of enteric, and true relapsing fever, however, are so entirely different, that it is impossible for any person practically acquainted with them, to mistake the one for the other (see pages 290, 385).

3. *Remittent Fever.* The diagnosis between pythogenic and remittent fever is often extremely difficult. The remitting character of pythogenic fever has been already pointed out (pp. 476, 497). Trousseau,[t] and other observers,[u] record cases, where, at first, it put on an intermittent type; and they add, that this intermittent form is most common in malarious countries. It is highly probable that, in the tropics, many of the cases, reported as remittent fever, are really examples of pythogenic fever. This is the only way in which we can account for the circumstance that, until within the last few years, pythogenic fever was said to be unknown in India.[x] Such mistakes are not surprising, for vomiting and diarrhœa may occur in remittent fever; while enlargement of the spleen, cerebral symptoms, and the typhoid state, are common to both diseases. The eruption of lenticular spots is, perhaps, the only reliable distinctive mark of pythogenic fever; and, in every case of remittent fever, complicated with abdominal symptoms, they ought to be carefully looked for.

4. *Scarlatina.* Cases of pythogenic fever, in which the lenticular spots are preceded by a uniform scarlet rash, are apt to be mistaken, at first, for scarlatina, especially if there be, at the same time, sore-throat (see page 473). As a rule, however, there is no sore-throat in these cases; the tongue is not like that of scarlet fever; and the scarlet rash does not make its appearance before the fourth or fifth day of the disease. The pulse, also, is usually slower, and the temperature less, than at the corresponding stage of scarlet fever.

5. *Variola.* I have known a copious eruption of lenticular spots mistaken for the papules of variola. Lenticular spots, however, are never so prominent, or so hard, as the papulæ of smallpox; they do not suppurate; and they rarely appear before the seventh day of the disease. Moreover, the severe lumbar pain, and the greater severity of the primary symptoms observed in

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[t] TROUSSEAU, 1861, p. 171. [u] BARTLETT. 1856, p. 134. See also *antea*, p. 497.
[x] See MOREHEAD, *Dis. of India*, 1st ed. i. 307.

variola, contrast strongly with the mode of accession of pythogenic fever.

6. *Pyæmia* may simulate pythogenic fever very closely. The absence of lenticular spots, the yellowish tint of countenance, and the circumstances under which the symptoms appear, usually suffice to distinguish the former malady. Many cases of *Puerperal Fever* put on all the symptoms of pythogenic fever. Although, occasionally, it may be impossible to form a positive diagnosis between the two affections, the case may be assumed to be puerperal fever, if there be no lenticular spots, if no other case of pythogenic fever has occurred in the same house, and if the symptoms have commenced within two or three weeks of confinement.

7. *Pneumonia.* Latent pneumonia is apt to be mistaken for pythogenic fever. In children, pneumonia is often accompanied by great sympathetic disturbance of the stomach and bowels, which obscures the primary disease;[y] while in adults, as Dr. Bristowe has pointed out, pneumonia is not unfrequently complicated with acute dysentery.[z] On the other hand, pythogenic fever may be complicated with pneumonia. The physical signs of pneumonia being present, lenticular spots, even in the absence of diarrhœa, would indicate pythogenic fever complicated with pneumonia; but, failing spots, the symptoms will, in most cases, be correctly referred to pneumonia, especially if no other cases of fever have occurred in the same house, and if the signs of pneumonia have been discovered within the first ten days of the illness (see page 214).

8. *Acute Phthisis* is often very apt to be mistaken for pythogenic fever. A quick pulse, perspirations, great emaciation, circumscribed flushes on the cheeks, a dry tongue, great muscular prostration, delirium, stupor, dyspnœa, and bronchitic râles are phenomena common to both affections. Pulmonary consolidation may be due to the secondary pneumonia of pythogenic fever; and in phthisis, diarrhœa may result from tubercular ulcers of the bowel. In phthisis, however, the belly is usually depressed, in place of being distended and tympanitic, as in fever. The presence of lenticular spots is positive evidence in favour of fever; but, unfortunately, their absence does not prove the contrary. In all doubtful cases, the family history, and the circumstances under which the disease has appeared, ought to be investigated; and the fact that acute phthisis may be developed, as a complication or

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[y] West, 1848, ed. 1854, p. 266; Barthez and Rilliet, 1853, ii. 699.
[z] *Trans. Path. Soc.* viii. 66.

sequela of pythogenic fever, must not be lost sight of (see page 503).

9. *Tubercular Meningitis.* Greater difficulty is often experienced in making a diagnosis between tubercular meningitis and pythogenic fever, than perhaps between any other known diseases. Many writers have laid down rules for distinguishing them;[a] but at the bed-side, all these rules are too often unavailing. A quick pulse, headache, delirium, vomiting, occasional remissions, and even partial palsy, are met with in both diseases. Lenticular spots, which, when present, would at once settle the matter, are unfortunately least abundant, and oftenest absent, at the period of life when the difficulty in diagnosis is likely to arise. In the absence of eruption, the following are the points of distinction most to be relied on:—In tubercular meningitis, the vomiting at the outset is usually more urgent; the tongue is rarely dry and brown, as it is in most cases of fever with severe cerebral symptoms; the bowels are usually constipated, or, if there be diarrhœa, the stools are not ochrey-yellow, as in fever; the abdomen is usually depressed and painless, instead of being tender and tympanitic, as in fever; enlargement of the spleen, intestinal hæmorrhage, and epistaxis, often observed in fever, are not met with in meningitis; the headache is more severe than in fever, and may accompany, or alternate with, the delirium (see page 487), or be marked by the *cri hydrencéphalique;* partial paralysis points to meningitis, rather than to fever; and, lastly, the patient is more irritable, is more intolerant of light and sound, and offers greater resistance to any examination, in meningitis, than in fever. The occurrence of other cases of fever, in the same house, would favour the supposition of fever; while the circumstance of other children, in the same family, having died of hydrocephalus, would support an opposite conclusion.

10. *Cerebral Softening.* When stupor and other cerebral symptoms supervene, within the first few days of pythogenic fever, and are associated with vomiting, the case may be mistaken for cerebral softening; and Louis mentions a case of cerebral softening, which was thought to be pythogenic fever.[b] Most of the points of distinction enumerated under the head of tubercular meningitis, are equally applicable here (see also page 213).

11. *Gastro-enteritis.* Under this term, may be included all those derangements of the stomach and bowels, such as ordinary

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[a] Watson, *Lect. on Pract. of Phys.* 1848, i. 434; West, 1848, ed. 1854, p. 67.
[b] Louis, 1841, ii. 189.

diarrhœa, enteritis, colitis, and the *embarras gastrique* of French writers, in which the febrile symptoms are secondary, instead of being primary. In adults, these acute affections are usually distinguished without difficulty from pythogenic fever, by the absence of eruption, and by headache, cerebral symptoms, epistaxis, and enlargement of the spleen; by the comparatively small amount of fever, or of muscular prostration; and by the characters of the stools, which do not present the ochrey-yellow appearance of those of pythogenic fever. The reader is referred to a table, in which Louis compares the symptoms of 17 cases of fever with 23 of enteritis.[c] Dysentery is easily distinguished by the tenesmus, and by the peculiar characters of the stools. In the case of children, however, from two to five years of age, in whom general disturbance and delirium are much more apt to result from local causes than in adults, the diagnosis is occasionally extremely difficult, if not impossible.[d] The presence of rose-spots, however, always settles the question.

12. *Peritonitis.* I have met with one or two cases of tubercular peritonitis, which closely resembled pythogenic fever. The symptoms were: fever, with occasional perspirations, vomiting, abdominal pain and tenderness, diarrhœa, great prostration and emaciation, circumscribed flushes on the cheeks, bronchitic râles, and ultimately slight delirium at night. In most cases, however, of tubercular peritonitis, the abdomen is retracted, and the bowels are confined.

Section XI.—Prognosis and Mortality.

Prognosis in pythogenic fever is based on the rate of mortality; the circumstances influencing the rate of mortality; the presence and severity of certain symptoms and complications in individual cases; and the mode of fatal termination.

a. Rate of Mortality.

Table XXXVII.[e] gives the rate of mortality among the cases of pythogenic fever, admitted into the London Fever Hospital, during 14½ years.

Thus, out of 2505 cases, 465 died, making a mortality of 18·56 per cent., or of 1 in 5·38; but deducting those patients, who were moribund on admission, the mortality falls to 17·2 per cent., or to 1 in 5·8. This rate of mortality is, on the whole, slightly less than that of typhus (see page 217).

The rate of mortality from pythogenic fever in the London Fever Hospital does not exceed that observed elsewhere, care being

[c] Louis, 1841, ii. 209. [d] See Barthez and Rilliet, 1853, ii. 699.
[e] See note, page 217.

taken to exclude other forms of fever, in making the calculation. Out of 137 cases admitted into Guy's Hospital in the two years, 1854 and 1861, 26 died, or 19 per cent.[f] Of 74 cases under Dr. Peacock, at St. Thomas's Hospital, 14, or 18·9 per cent., were

TABLE XXXVII.

Years.	Admissions.	Deaths.	Mortality per cent.
1848	152	41	26·97
1849	138	26	18·84
1850	137	24	17·51
1851	234	30	12·82
1852	140	25	17·85
1853	211	59	27·96
1854	228	44	19·3
1855	217	31	14·28
1856	149	23	15·43
1857	214	30	14·02
1858	180	27	15·
1859	176	33	18·75
1860	94	27	28·72
1861	161	32	19·87
1862 to June 30th	74	13	17·56
Total	2505	465	18·56
Deducting 18, who died within 24 hours	2487	447	17·99
Deducting 41, who died within 48 hours	2464	424	17·2

fatal.[g] Of 131 cases admitted into King's College Hospital during 18 years, 27, or 20·61 per cent. died.[h] Of 843 cases admitted into the Glasgow Infirmary in twelve years (1847-1853,[i] and 1857-1861[k]), 155, or 18·3 per cent., died. Of 190 cases under Forget, at Strasbourg, 44, or 23·15 per cent., died;[l] and of 147 cases under Chomel, at Paris, 47, or 32 per cent., died.[m] Again, of 9974 cases, reported as occurring in the provinces of France, between the years 1841 and 1846, 1667 died, or the mortality was 16·7 per cent.;[n] and of 4611 cases at Paris, in 1854, 1002, or 21·73 per cent., died.[o] Adding together all the above figures, we have 18,612 cases, and 3447 deaths, or the mortality is 18·52 per cent, or 1 in 5·4, a result which is almost identical with that obtained in typhus (see page 218).

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[f] WILKS, 1855; and *Report* for 1861. [g] PEACOCK, 1856 (No. 1).
[h] *Brit. & For. Med. Chir. Rev.* Oct. 1860, p. 332. [i] M'GHIE, 1855, p. 161.
[k] *Hosp. Reports.* [l] FORGET, 1841, p. 440. [m] CHOMEL, 1834.
[n] DE CLAUBRY, 1849, p. 31. [o] DAVENNE, 1854.

b. Circumstances influencing the Rate of Mortality.

1. *Times and Seasons.* Table XXXVIII. shows the rate of mortality, according to season, of the cases admitted into the London Fever Hospital during ten years (1848-57).

TABLE XXXVIII.

Seasons.	Admissions.	Deaths.	Mortality per cent.
Spring . . .	222	44	19·82
Summer . . .	355	64	18·03
Autumn . . .	746	136	18·23
Winter . . .	497	89	17·91
Total	1820	333	18·29

From this Table it appears, that the mortality was slightly greater in spring, when the disease was least prevalent. Chomel,[p] Forget,[q] and Bartlett,[r] have endeavoured to show that the mortality in France and America is almost double in the cold months of the year, what it is in the warm; but their conclusions are drawn from a very limited number of cases. Of the 1820 cases admitted into the London Fever Hospital in ten years, the mortality during the warm months (May to October) was 18·2 per cent., and during the rest of the year, 18·5 per cent.

As regards different years, the mortality at the London Fever Hospital was much more equable, than in the case of typhus. Thus, while in typhus, the mortality in one year was only 8·8 per cent., and in another, as high as 60 per cent. (see page 217); in pythogenic fever, it was in no year below 12·8 per cent., or higher than 28.7 per cent.

2. *Sex.* Table XXXIX. shows the mortality, according to sex, of the cases admitted into the London Fever Hospital during ten years (1848-57).

TABLE XXXIX.

	Admissions.	Deaths.	Mortality per cent.
Males	905	160	17·68
Females	915	173	18·89
Males and Females . .	1820	333	18·29

The mortality was somewhat greater in females, which is the reverse of what holds good in typhus. Similar observations have

[p] CHOMEL 1834. [q] FORGET, 1841, p. 410. [r] BARTLETT, 1856, p. 125.

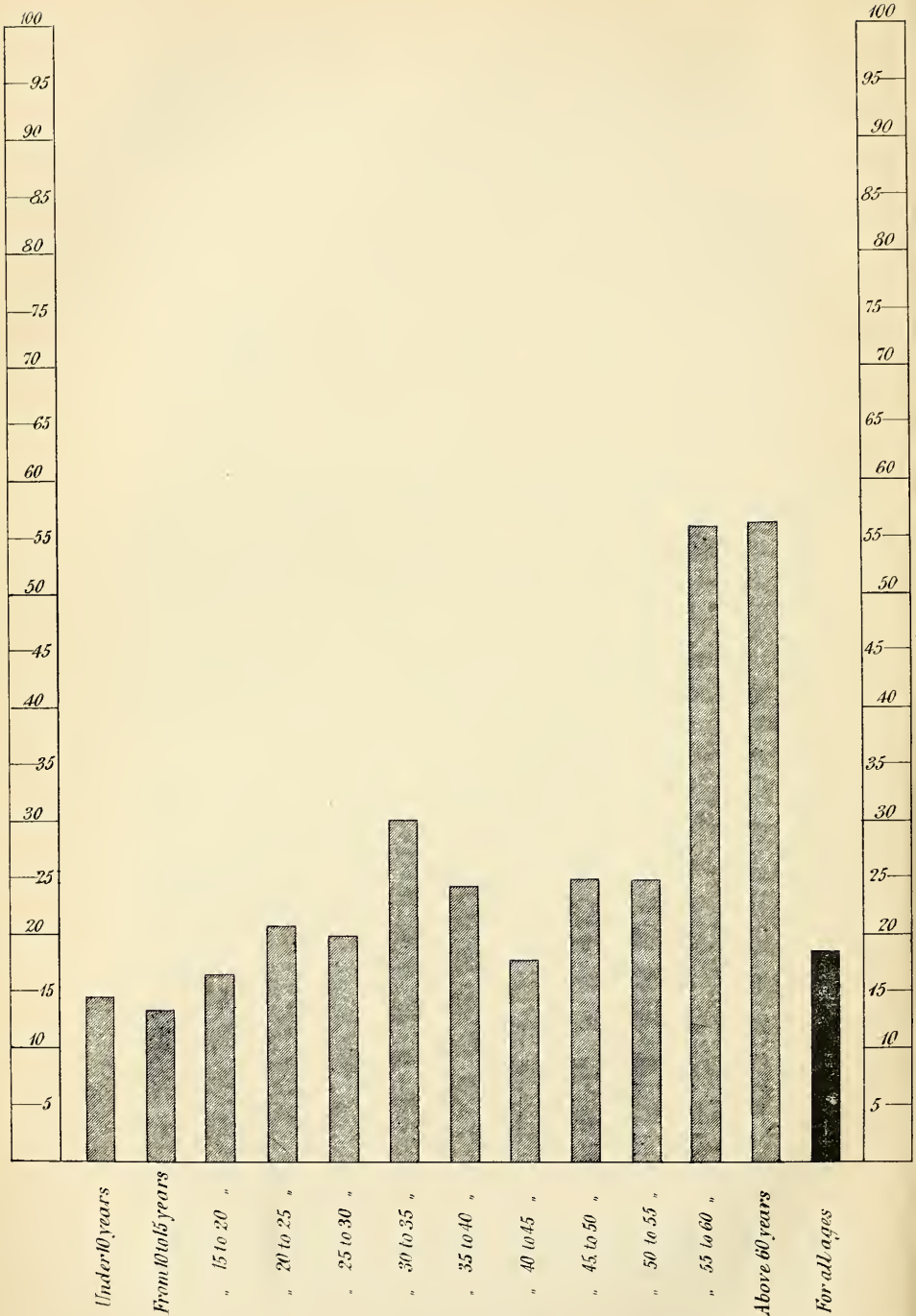

Diagram X, shows the variations according to Age, in the rate of Mortality, of 1772 cases of Pythogenic Fever, admitted into the London Fever Hospital. (Compare with Diag. V.)

been made on the continent. Of 1687 male cases in the provinces of France, 227, or 13·4 per cent. died; while of 2307 females, 336, or 14·5 per cent. died.[s] The statistics given by Forget[t] and Chomel,[u] although on a smaller scale, also make the mortality greater in females: and according to Friedreich[x] and Friedleben,[y] the mortality is greater in girls than in boys. An opposite observation has been made at Glasgow. Of 270 males admitted into the infirmary, from 1857 to 1861, 48, or 17·7 per cent., died; whereas, of 217 females, only 30, or 13·8 per cent., died.[z] The greater mortality in females may be partly accounted for by the existence of pregnancy as a complication (see page 515).

3. *Age.* Tables XL and XLI., show the influence of age on the mortality of the cases admitted into the London Fever Hospital, during ten years (1848-1857).

Table XL.

	Number	Mean Age.
Total admissions in which age known	1772	21·25
Cases which recovered . . .	1444	20·7
Cases which died	328	23·54

Table XLI.

Age.	No. of Cases.	Deaths.	Mortality per cent.	Age.	No. of Cases.	Deaths.	Mortality per cent.
				Brought forward..	1680	303	
Under 5 years ...	4	..	..	From 40 to 45 yrs.	46	8	17·39
From 5 to 10 years	103	15	14·43	„ 45 to 50 „	20	5	25·
„ 10 to 15 „	250	32	12·8	„ 50 to 55 „	8	2	25·
„ 15 to 20 „	519	84	16·18	„ 55 to 60 „	9	5	55·55
„ 20 to 25 „	404	82	20·3	„ 60 to 65 „	7	4	57·14
„ 25 to 30 „	240	46	19·16	„ 65 to 70 „	1	1	100·
„ 30 to 35 „	100	30	30·	„ 75 to 80 „	1	..	..
„ 35 to 40 „	60	14	23·33	Age not known ..	48	5	10·41
Carried forward	1680	303		Total	1820	333	18·29

It is obvious, that age does not exercise that remarkable influence on the rate of mortality, observed in typhus and relapsing fever (see pages 221 and 368). The difference will be at once apparent on comparing Diagrams X.[a] and VI. with V. and II.) There is a

[s] De Claubry, 1849, p. 31. [t] Forget, 1841, p. 403. [u] Chomel, 1834, p. 357.
[x] Friedreich, 1856. [y] *Brit. & For. Med. Chir. Rev.* July, 1858, p. 161.
[z] *Hosp. Reports.*

[a] The two columns, showing the mortality above 55, might have been omitted from Diagram X, as the total number of cases was extremely small.

greater uniformity in the rate of mortality at different periods of life in pythogenic fever, than in typhus, and the small rate of mortality observed in early life in typhus, does not hold good in pythogenic fever. Thus, in typhus, the rate of mortality between ten and twenty years of age was under five per cent.; but in pythogenic fever it is upwards of fifteen per cent. Again, although the mortality from pythogenic fever increases with the age, it does so to a less extent than in typhus. Thus, of those

Above 30 years of age 27·38 per cent. died.
„ 40 „ 27·17 „
„ 50 „ 46·15 „

As in typhus, the rate of mortality between forty and forty-five is considerably less than in the period of life immediately preceding, but here the reduced rate of mortality is in the male sex, in which it is as low as 14·28 per cent (see page 222).

Louis states, that none perished out of 6 of his patients under seventeen years, and he observes that during ten years' hospital experience, he had only known one case prove fatal under twenty.[b] Probably few cases were admitted into the Hotel Dieu, at an early age. Barthez and Rilliet ascertained that 29 out of 111 children attacked with 'typhoid fever' died.[c] Of 9974 cases observed throughout the provinces of France, 2282 were under fifteen years of age; and of these, 256, or 11·22 per cent., died: of 7692 above fifteen, 1411, or 18·34 per cent. died.[d] At Paris, in 1854, of 260 cases under fifteen, 68 (26·15 per cent.) died; of 4275 cases between fifteen and fifty, 911 (21·31 per cent.) died; and of 76 cases above fifty, 23 (30·26) died.[e]

4. *Station in Life.* Dividing the patients in the London Fever Hospital into three classes, viz.:—1. Paying patients; 2. Free patients, and those who have not been in the receipt of parish relief prior to their illness; and 3. Parochial paupers; the mortality of pythogenic fever, in each class, was as follows:—

TABLE XLII.

	No. of Cases.	Deaths.	Mortality per Cent.	Mortality per Cent. of Typhus.
First Class - - -	281	47	16·72	14·89
Second „ - - -	1454	273	18·77	18·6
Third „ - - -	85	13	15·3	27·64

The rate of mortality does not appear to be greater among the

[b] LOUIS, 1841, ii. p. 354. [c] BARTHEZ and RILLIET, 1853.
[d] DE CLAUBRY, 1849, p. 31. [e] DAVENNE, 1854.

destitute than in the better classes. In private practice, pythogenic fever is probably more fatal among the upper classes, than among the very poor. Chomel[f] and Forget[g] both regard debility from destitution, a favourable circumstance, as regards prognosis. Pythogenic fever is as prevalent and as fatal among the rich, as among the poor; typhus is not only most prevalent, but most mortal, among the very poor (see page 222).

5. *Recent Residence in a Locality.* Of 1787 patients affected with pythogenic fever, who had resided in London more than six months prior to their admission into the Fever Hospital, 279, or 15·61 per cent., died; whereas of 191 patients, who had resided in London less than six months, 37, or 19·37 per cent., died. The difference was not accounted for by any difference in age. Of 68 patients under Louis and Chomel, who had resided in Paris less than six months, 27, or 39·7 per cent., died; whereas of 151 patients, who had resided a longer time, 46, or 30·46 died.[h] As far as these figures go, they show that recent residence in an infected locality increases the fatality of pythogenic fever (see page 223).

6. *Place of Birth and Race.* Of the patients admitted into the London Fever Hospital during fourteen years, the mortality, according to birth-place, was as follows:—

TABLE XLIII.

	No. of Cases.	Deaths.	Mortality per cent.
English - - - - -	1813	318	17·54
Irish - - - - - -	162	15	9·26
Scotch - - - - -	13	2	15·38
Foreigners - - - -	17	3	17·64
Birth-place not noted -	426	114	26·76

The small rate of mortality among the Irish is remarkable. The large mortality among the patients, whose birth-place was not noted, admits of the same explanation as in typhus (see page 223).

7. *Intensity of the Poison and Family Constitution.* Several deaths often occur in the same family from pythogenic fever, whereas all the members of other families, attacked at the same time, often recover. This circumstance may be partly due to family constitution, for occasionally several members of the same family die from pythogenic fever, at distant places, and at long intervals. This explanation, however, is not sufficient; for the remark applies

[f] CHOMEL, 1834. [g] FORGET, 1841, p. 404.
[h] LOUIS, 1841, ii. p. 357; CHOMEL, 1834, p. 358.

to members of different families residing in the same house: while in country-districts, it is often found that the mortality is much greater in one village, than in another a few miles off. Such observations point to differences in the intensity of the poison.

8. *Debility*, from previous diseases, or from any other cause, has not the same unfavourable effect on the mortality of pythogenic fever, as it has on that of typhus. On the contrary, it has been a common observation, on the part of almost every writer, who has paid attention to the subject, that the strong and robust succumb more readily to pythogenic fever, than the feeble[1] (see page 224).

c. Presence of certain Symptoms and Complications.

With a few exceptions mentioned below, the rules for prognosis laid down under typhus (page 225), hold good in pythogenic fever. A few rules may be added here, which apply to pythogenic fever alone.

1. It ought always to be kept in mind that the disease is often latent, and that the mildest cases may terminate suddenly in death (page 520).

2. A fall in the pulse is a less favourable indication than in typhus, as the frequency of the pulse may vary greatly, before the cessation of the fever.

3. Many more patients die, in whom the tongue is at no time dry and brown, than in the case of typhus.

4. Vomiting, early in the attack, is a favourable symptom, rather than otherwise; but appearing suddenly after the fourteenth day, it should put us on our guard against peritonitis.

5. Diarrhœa is an unfavourable symptom, in proportion to its severity and duration.

6. Abdominal pain and great meteorism are also unfavourable.

7. Hæmorrhage from the bowels is always unfavourable; when slight, it adds little to the danger; but when once it occurs, it may suddenly become profuse.

8. Epistaxis is, in most cases, of little moment; but if profuse, it may be fatal.

9. An abundant eruption does not betoken a grave case, as in typhus (see page 471).

10. When the skin is very hot at the commencement, the subsequent course of the disease is often severe.

11. Perspirations may occur at any stage of the fever, and do not necessarily indicate an approaching termination.

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[1] Chomel, 1834; Forget, 1841, p. 404; Barrallier. 1861, p. 282.

12. When peritonitis supervenes, the case is almost hopeless; but patients have occasionally recovered after all the symptoms of peritonitis from perforation.

13. Severe muscular tremors, in cases where the mind is clear, indicate deep and rapid ulceration of the bowel.

14. Extreme prostration, coming on suddenly, should lead to the suspicion of hæmorrhage into the bowel, or of perforation, even if there be no external pain.

15. Pregnancy is always a very serious complication (page 515).

16. A temporary remission, during the second or third week of the disease, followed by a return of all the symptoms in an aggravated form, usually terminates in death. Louis and Chomel[k] make a similar remark.

17. Even after convalescence has commenced, all cause for anxiety is not removed. A relapse may occur, or the intestinal ulcers, instead of cicatrizing, may give rise to exhausting diarrhœa, or advance to perforation.

d. Mode of Fatal Termination.

In many cases of pythogenic fever, death takes place by asthenia, which may be ascribed to a morbid condition of the muscular tissue of the heart, to the profuse character of the diarrhœa, or to the protracted interstitial death of the tissues, resulting from the febrile process. Along with this asthenia, there may be a varying degree of anæmia, arising from inanition, from intestinal hæmorrhage, or from epistaxis. Although death takes place by pure asthenia or anæmia more commonly in pythogenic fever than in typhus, yet, in most cases, it is preceded by more or less coma, owing to the accumulation in the blood of the products of the destruction of the tissues. In a large number of cases, death is directly due to some complication, such as peritonitis, pneumonia, or erysipelas.

Section XII.—Anatomical Lesions.

The anatomical lesions of few diseases have been studied with greater care, than those of pythogenic fever. My own observations, on upwards of fifty fatal cases, confirm those contained in the classical works of Louis, Chomel, Rokitansky, and Jenner.[l]

Pythogenic fever differs from typhus and relapsing fever, in the invariable presence of specific lesions, which are often associated with others of an accidental or less constant character.

[k] Louis, 1841, ii. 349; Chomel, 1834.

[l] Louis, 1841; Chomel, 1834; Rokitansky, *Path. Anat.* Syd. Soc. Transl. ii. 68; Jenner, 1849 (2).

a. Generalities.

1. *The Cadaveric Rigidity* is more marked, and of longer duration than in typhus. Of 10 cases, where I have noted the circumstance within thirty-six hours after death, there was marked rigidity in all but 1.

2. *Emaciation.* Owing to the lengthened duration of the illness, the emaciation is often extreme. There is always considerable emaciation, when the disease has lasted longer than three weeks.

3. *Putrefaction.* On the whole, there is less tendency to rapid putrefaction of the dead body than in typhus, except in cases where the typhoid state has existed for some days prior to death.

b. Integuments and Muscles.

1. *Discolourations.* Livid discolouration of the integuments on the dependent parts of the body is, as a rule, less extensive than in typhus, and rarely extends up along the sides of the trunk, when the body has been laid on the back. The face is rarely livid, except where there have been pulmonary complications. Discolouration of the integuments, along the course of the sub-cutaneous veins, is scarcely ever observed. Greenish discolouration of the integuments covering the abdomen, within forty-eight hours after death, is also rarer than in typhus. Louis and Jenner noted this appearance in only 6 of 46 cases (see page 229).

2. *The Eruption.* The lenticular rose-spots are never observed on the dead body, although they may have been present in large numbers immediately before death. (See page 515).

3. *Sudamina* are not uncommon. Jenner noted them in 4 out of 23 cases (see page 474).

4. *Erysipelas* (see page 516).

5. *Bed-sores and Gangrene* (see page 516).

6. *The Muscles* rarely present the unusually dark colour and soft consistence, so common after death from typhus. Louis states that, with the exception of the heart, he always found them normal, both in colour and in consistence.[l] Jenner noted the muscles as abnormally dark in only 1 of 23 cases.

c. Organs of Digestion.

1. *Pharynx and Œsophagus.* The pharynx is, in many cases, found to be healthy (in 38 of 46 cases by Louis, and in 7 of 15 cases by Jenner), but not unfrequently it exhibits signs of recent inflammation, and sometimes distinct ulcers. Louis found recent ulcers in 6 out of 46 cases,[m] and Jenner in 5 out of 15 cases. These

[l] Louis, 1841, i. 378. [m] Ibid. i. 135.

ulcers are seated chiefly at the lower part of the pharynx; they have a round, oval, or irregular outline, and they vary in diameter from two lines to three-quarters of an inch. They are usually very superficial, but occasionally their base is formed by the muscular coat. Their edges are not thickened, and the surrounding mucous membrane is either normal or slightly injected. In cases where there is no ulceration, the mucous membrane is occasionally found to be abnormally injected, or coated with diphtheritic false membrane, or the sub-mucous tissue is infiltrated with serum or pus.

The œsophagus is in most cases healthy; but it occasionally exhibits ulcers, similar to those met with in the pharynx (in 7 of 46 cases Louis; in 1 of 15, Jenner). These ulcers present the same characters as those of the pharynx, and are usually largest and most numerous at the lower, or cardiac, extremity. They may be mere excoriations, or they may penetrate to the muscular coat, but they have never been found to end in perforation.

The ulcers in the pharynx and œsophagus are never found when death occurs before the third week of the disease. Although they are not met with after death from typhus, or from other acute diseases, they must not be confounded with the specific lesions of pythogenic fever, to which they are secondary. There is no evidence that they are preceded by any morbid deposit like what occurs in the intestines, although statements to this effect are commonly made. Chomel remarks:—'Toutes les ulcérations, dont 'nous avons parlé jusqu'ici, succèdent à une altération des folli-'cules: dans celles dont nous occupons maintenant, cette altération 'n'a point été constatée; on n'a jamais rien observé dans ces 'parties d'analogue aux plaques gauffrées ou aux follicules isolés 'engorgés de l'intestin.'[n] Louis expresses himself in almost the same terms.[o]

2. *The Stomach* is, in many cases, healthy. The morbid appearances, which it sometimes presents, are increased vascularity, softening, mammillation, and superficial ulcers. These lesions, however, are far from constant, and are observed with almost equal frequency after death from other diseases. Louis pointed out, long ago, that 'typhoid fever' has no more right to be designated *gastro-entérite*, than pneumonia has to be called *gastro-peripneumonie*.

Increased vascularity was noted by Jenner, in 5 out of 15 cases; but in 6 cases the membrane was pale. Chomel found the mucous membrane of the stomach, in some cases, pale throughout.

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[n] Chomel. 1834, p. 192. [o] Louis, 1841, i. 136.

Softening of the mucous membrane was noted by Louis in 16 of 46 cases; by Chomel in 14 of 42 cases; and by Jenner in 5 of 15 cases. This softening is, in most cases, confined to the great *cul de sac*, but it is occasionally general. Sometimes, the membrane is attenuated, as well as softened. In two cases, Chomel found the membrane entirely destroyed, fragments only remaining, which were readily washed off by a stream of water, while, in a third case, the softening had extended through the entire coats, over a space the size of a half-crown piece, so that very slight pressure caused it to rupture. This softening of the coats of the stomach is probably nothing more than the result of *post-mortem* digestion. Chomel showed that there was no relation between it and the presence of gastric symptoms during life.

Mammillation of the mucous membrane was observed by Louis in 13 out of 46 cases, and by Jenner in 6 out of 8 cases.

Ulceration is extremely rare. I have never met with it in upwards of 20 cases, in which I have examined the stomach. Chomel failed to find it in any of 42 cases examined by him. Jenner met with it only once in 20 cases; and Louis, four times in 46 cases. The ulcers are not confined to any particular part of the stomach. They may exceed twenty in number, but they are always minute, varying from the size of a pin's head to two or three lines in diameter. They are also quite superficial, and are not preceded by any deposit in the mucous membrane. These minute superficial ulcers of the stomach are not uncommon after death from various diseases. I have often met with them in the bodies of patients who have died of chronic diseases of the heart or liver, and Dr. G. Budd says that he has several times had reason to ascribe them 'to an excessive use of stimulants, given in the hope 'of remedying the sense of sinking in the last days of life'[p] (see page 230).

3. *The Duodenum* is, in most cases, healthy. Sometimes, its lining membrane appears abnormally vascular, or its mucous follicles are enlarged; but these conditions were ascertained by Louis to be equally common after death from other acute diseases. In 2 out of 22 cases, Louis found one or two minute superficial ulcers, close to the pylorus, similar to those met with in the stomach. There was no ulceration in any of 15 cases examined by Jenner, or in 20 cases dissected by myself.

4. *The Jejunum and Ileum* do not usually contain much gas. On the contrary, the lower part of the ileum is often collapsed and

[p] Budd, *Dis. of Stomach*, 1855, p. 153.

empty. The tympanitis, during life, is mainly due to the presence of gas in the colon, except in cases where there is peritonitis, when the intestines may be uniformly distended. Excluding cases of perforation, Louis found slight tympanitis of the small intestine in only 14 out of 39 cases. The fæcal contents are liquid, and of an ochrey or orange colour, and they often contain yellowish-brown sloughs detached from the mucous membrane, large quantities of ammoniaco-magnesian phosphate, and occasionally small masses of blood. A considerable quantity of intestinal mucus may be found in the upper part of the small intestine. Louis states that worms (*ascaris lumbricoides*) are often passed from the bowel by patients labouring under pythogenic fever, and that he has, on many occasions, found them in the small intestine after death. Although the presence of worms in the intestines is, in my experience, exceptional, the observation is interesting in connexion with some of the names applied to pythogenic fever by certain writers (see pages 387 and 390).

In 3 out of 46 cases, Louis found the upper portion of the small intestine invaginated in the portion below, to the extent of from one to two feet (see also Case XXVIII., page 464). These invaginations are not accompanied by any signs of inflammation, and are not uncommonly produced in the death-struggle of many diseases, in which there is much torpor of the cerebro-spinal system.

The colour of the mucous membrane varies. It is a mistake to imagine that its vascularity is necessarily increased. Louis observed it of its natural paleness, or merely tinged with bile, in 17 out of 46 cases; and Jenner, in 11 out of 17 cases. This fact is important, as bearing on the doctrine which ascribes the symptoms of pythogenic fever to enteritis. In one-third of Louis' 46 cases, there was increased redness of the mucous membrane. This redness may be either uniform, or in patches, and it is almost always most marked towards the cæcal extremity. When death does not occur until after the third or fourth week of the disease, the mucous membrane is often found to present a greyish or slate-coloured aspect, an appearance which was attributed by Louis to a transformation of the redness, occasionally observed at an earlier stage. Chomel found the mucous membrane of the small intestine infiltrated with a bloody fluid, over a space varying from four inches to three feet, in 7 out of 42 cases. The membrane was much increased in thickness, and presented a gelatinous glistening aspect, and a rose- or reddish-black colour. When squeezed, a bloody fluid oozed out, and the membrane regained its natural thickness. This appear-

ance was uniform all round the bowel, and was not limited to the dependent portions of the coils. In most of the cases, there had been intestinal hæmorrhage during life, or blood was found in the intestines after death. I have met with a similar condition in one case.

As to consistence, Louis found the mucous membrane softened in all except 9 of 42 cases. Chomel noted this appearance in only 5 of 42 cases; and Jenner, in 3 of 15 cases. This softening, like that in the stomach, is probably a *post-mortem* change,[q] although Louis was inclined to think that in certain cases, where it was associated with redness and thickening, it was inflammatory.

None of the above lesions are constant in, or peculiar to, pythogenic fever (see page 231). The specific lesions, which are invariably present, and which consist in a peculiar disease of the agminated or of the solitary glands of the ileum, have now to be described.

These lesions present different appearances, according to the duration of the illness prior to death. They may be described as passing through four distinct stages, namely:—1. The stage of enlargement, or of deposition in the intestinal glands. 2. The stage of softening and ulceration. 3. The stage of the genuine pythogenic ulcer; and 4. The stage of cicatrization. Two or more of these stages may often be traced in the same body; for the morbid process, as well as the process of reparation, always commences at the extremity of the ileum nearest the cæcum, and proceeds upwards.

a. First Stage. This consists in the deposition of morbid material in and about the agminated and solitary glands. Rokitansky maintains that the deposition in the glands is preceded by a 'congestive stage; and Trousseau states that deposition does not commence before the fourth or fifth day of the disease.[r] There is no evidence, however, that the enlargement of the glands is preceded by increased vascularity; while it is not improbable that the enlargement commences with the disease, and is the cause, rather than the consequence, of the increased vascularity. In no case, where death has occurred at an early stage of the disease, has there been increased vascularity without enlargement of the glands; and, indeed, in no case has the latter appearance been wanting. In one case under my care, where death occurred on the sixth day, great enlargement had already taken place (see

[q] See Budd, *Dis. of Stomach*, 1855, p. 46.

[r] Trousseau, 1861, p. 139.

Case XXVII). Considerable deposition had also taken place in four cases recorded by Brétonneau,[s] Forget,[t] and Bristowe,[u] where death took place on the fifth day. In the case mentioned at page 439, where death occurred at the end of the second day, there was also considerable enlargement. The appearances presented by the mucous membrane of the lower part of the ileum, in this case, are represented in the annexed wood-cut. Lastly, in the cases which occurred at Clapham, in 1829 (see page 438) considerable enlargement was found at the end of the first day. Moreover, in examining the agminated glands at the uppermost limit of the disease, in a case proving fatal at a more advanced stage, it is not found, as a rule, that there is increased vascularity without deposition; while, on the other hand, slight enlargement, without any increase of vascularity, is not uncommon. At all events, mere increased vascularity of the agminated and solitary glands, without any enlargement, will not justify the opinion that a patient has died of enteric fever, however short may have been the duration of the illness.

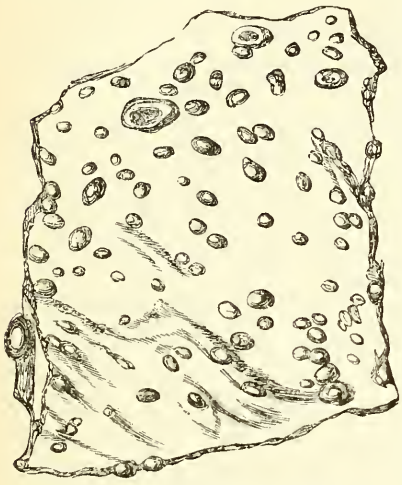

Fig. 6.—Lower three inches of ileum from a case of pythogenic fata¬ at the end of the second day

It is very rare that an opportunity is afforded of examining the intestines, before the eighth or tenth day of the disease. Peyer's patches are then found to be indurated, and elevated from half-a-line to two lines above the level of the mucous membrane. The membrane covering them is of a pinkish-grey or purplish colour, and it is often softened. The mucous membrane between the diseased patches may have its natural hue, or may present every grade of vascularity up to the most intense injection. The peritoneum corresponding to the patches, is usually much injected, and of a bright-red or pale-purple colour. Two varieties of diseased patches have been described by most French writers since the time of Louis. These are the *plaques molles* and the *plaques dures* of Louis; or the *plaques réticulées* and *plaques gauffrées* of Chomel. In

[s] Bretonneau, 1829. p. 70. [t] Forget, 1841, p. 119.
[u] *Lancet*, Ap. 28th, 1860; and *Path. Soc.* Jan. 7th, 1862.

the former, the mucous membrane covering the patch has a rugose or granular aspect, owing to the depressions between the glandules, which naturally exist on the patch, being enlarged, and the sub-mucous tissue is thickened and of a pinkish hue. In the latter, the patch is thicker and harder, the super-imposed mucous membrane presents a smoother and more uniform aspect, and the sub-mucous tissue contains a layer of yellowish-white, opaque, friable deposit. Louis was of opinion, that the *plaques dures* were less common than the *plaques molles*. He found them in only 13 of 46 fatal cases, and, from the circumstance that they were most common in cases fatal before the fifteenth day, he came to the conclusion, that this was a more dangerous form of the disease than the other. The correctness of this opinion may be doubted. At an advanced stage of the disease, after the morbid material has sloughed out, it is often impossible to say which of the two forms of the lesion has existed at first. From my own observations, I am inclined to think that the *plaques dures* constitute the more common form in adults. Moreover, the differences between the two forms are differences of degree, rather than of nature. Gradations may be observed between them, and they occasionally coexist in the same intestine. The morbid process is essentially the same in both (Fig. 7).

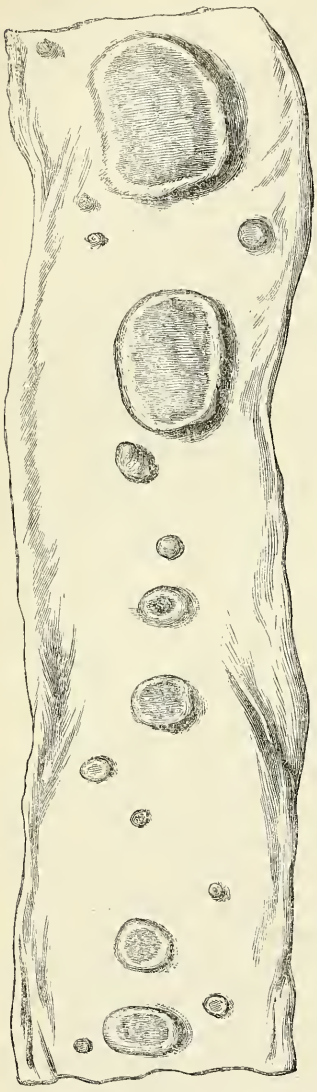

Fig. 7.—Portion of the ileum, from a case of pythogenic fever fatal on the tenth day, showing the enlarged agminated and solitary glands, not yet ulcerated.

The solitary glands at the lower end of the ileum are often affected in a manner similar to Peyer's patches. Louis found them diseased in 12 out of 46 cases. And, according to my experience, the proportion of cases, in which they are implicated, is even greater.

The solitary glands may be as large as a hemp-seed, or a split-pea, or they may be larger, and the white material in their interior often imparts to them an appearance not unlike pustules. The diseased solitary glands are usually limited to the lower twelve inches of the ileum, but they may extend higher. In exceptional cases, of which two are recorded by Chomel, the solitary glands are diseased, and Peyer's patches remain intact.

The precise situation of the deposit in the intestines is a point of some interest, on which different opinions have been expressed. Bœhm, in his admirable description of the intestinal glands,[x] stated that the matter was deposited in the submucous tissue external to the glandular sacs, and this is the opinion commonly taught. John Goodsir, however, after carefully investigating the morbid structure, pointed out that the matter is, *in the first place*, deposited in the interior of the glandulæ, which become much distended, and ultimately burst and discharge their contents into the submucous tissue.[y] Goodsir's statements were confirmed by Dr. Waters,[z] and agree entirely with my own examinations. The process is best traced in the solitary glands, but even in the agminated glands, the distended glandules may be observed at a sufficiently early stage.[a] Here, in fact, probably lies the real difference between the *plaques dures*, and the *plaques molles*. In the latter, the deposit is comparatively slight, and is confined to the glandules, the enlargement of which accounts for the rugose character of the patch; in the former, the glandules burst, and discharge their contents into the sub-mucous tissue, and consequently the surface of the patch becomes smoother. The manner in which these changes are effected will be readily understood from the annexed wood-cut, showing the structure of a Peyer's patch in its healthy condition. (Fig. 8).

The matter deposited in the intestinal glands does not, of necessity, lead to ulceration. There are good reasons for believing that it is occasionally re-absorbed.[b] According to Trousseau, resolution, independently of ulceration, may commence about the tenth day, and by the end of the third week, it may be complete. Thus we may account for those cases, already alluded to, where the

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[x] *De Gland. Intestin.* Berol. 1835. [y] GOODSIR, 1842. [z] WATERS, 1847.

[a] Goodsir found that when the still-adherent slough was gently raised under water, it sent processes down into the submucous tissue, which corresponded to the altered vesicles of the patch; and that after its separation, the ulcer exhibited a number of little pits, also corresponding to the enlarged glandules.

[b] On this subject, see TROUSSEAU, 1861, p. 139; CHOMEL, 1834, Obs. 14 & 15; LOUIS, 1841, i. 181; LYONS, 1861, p. 243; BARTHEZ and RILLIET, 1853, ii. 667.

disease is mild and of short duration (see pages 499 and 519). This favourable termination is probably restricted to the *plaques molles;*

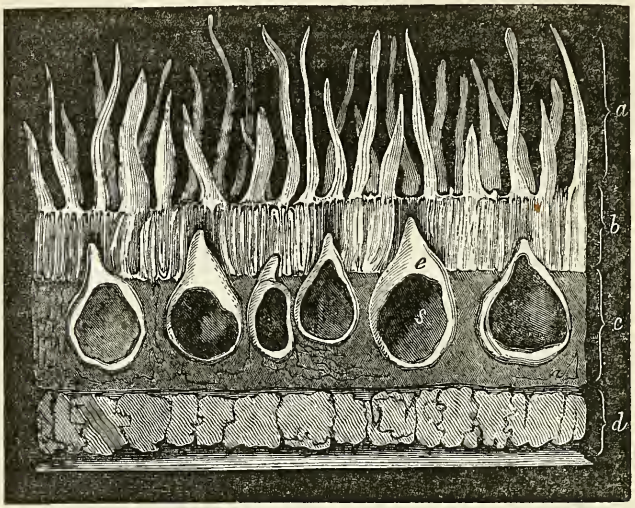

Fig. 8.—Vertical section through a Peyer's patch in a dog. *a.* Villi. *b.* Tubes of Lieberkühn, with the apices of Peyer's glands. *c.* Sub-mucous tissue with the glands of Peyer imbedded in it. *d.* Muscular and peritoneal coats. *e.* Apex of one of Peyer's glands. *f.* Peyer's gland laid open by the section. (TODD and BOWMAN).

after the glandules have discharged their contents into the sub-mucous tissue, it may be assumed that ulceration is inevitable.

b. Second Stage. Ulceration of the diseased Peyer's patches may commence in two ways. The mucous membrane may become softened, and one or more superficial abrasions may appear on the surface of the diseased patch, which may extend and unite into one large ulcer, and this ulcer may proceed to various depths through the coats of the bowel, and even to complete perforation. This is what happens in the case of the *plaques molles.* In the case of the *plaques dures,* the whole of the morbid material in the sub-mucous tissue, as well as the super-imposed mucous membrane, becomes detached in the form of a slough, leaving behind an ulcerated surface. This, according to my observation, is far the more common mode. The whole of a diseased patch may slough out at once, or it may slough in successive portions. Occasionally, the sloughing appears to extend at once through the entire coats of the intestine, so as to produce perforation. When death occurs between the twelfth and twenty-first days, the sloughs may usually be seen loosely attached to the intestinal ulcers, as represented in the annexed figure (Fig. 9). These sloughs have usually a peculiar yellowish-brown colour, from the imbibition of bile; occasionally they present

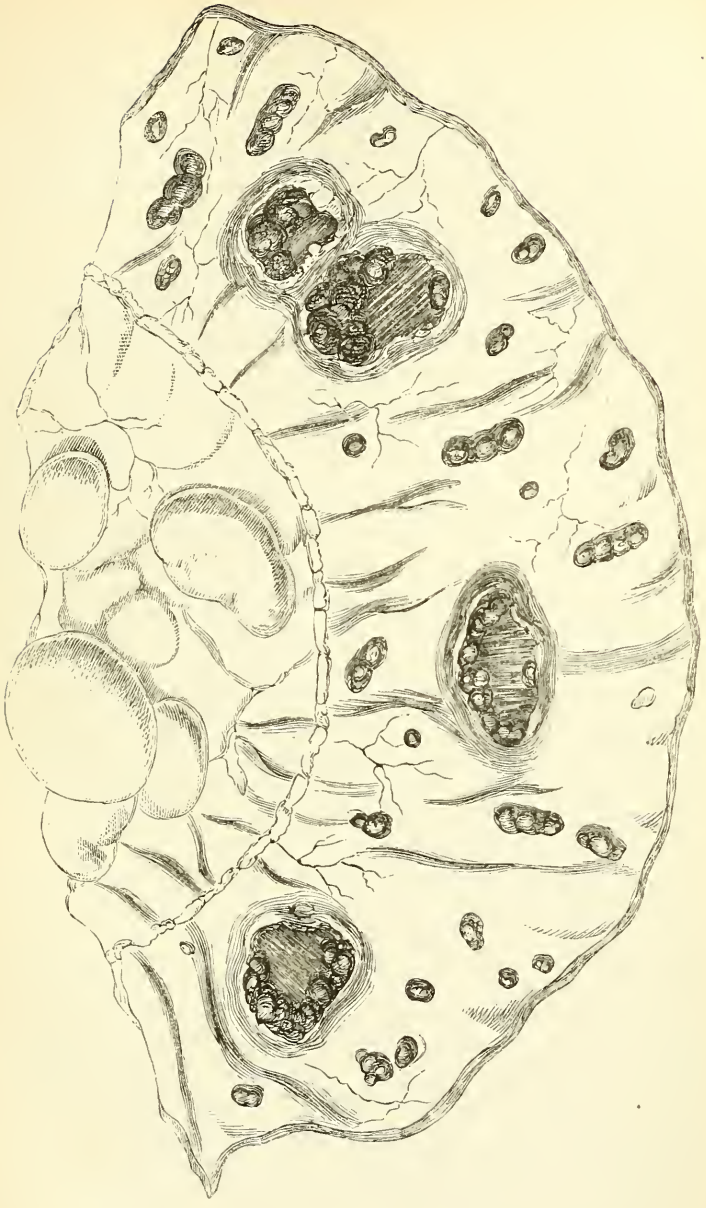

Fig. 9.—Portion of ileum, from a case of pythogenic fever, fatal on the 17th day, showing the partially detached sloughs. The morbid process has advanced further in the agminated, than in the solitary glands. The mesenteric glands are much enlarged.

a dark, spongy, fungating aspect, from being infiltrated with blood.

It is important to determine, at what stage ulceration commences. As a rule, this appears to be about the ninth or tenth day. Louis, Chomel, and Forget, record cases where death occurred on the eighth day, but in none had ulceration commenced, although in Forget's case, the agminated glands are described as on the point of ulcerating.[c] On the other hand, there are several cases on record, where ulceration has been found as early as the ninth or tenth day.[d] Ulceration, however, may commence earlier or later than the date here specified. Louis mentions two cases where it had commenced on the eighth day, and in one of Forget's cases, it was found, on the ninth day, to be very extensive. Cases have been already referred to (page 508), where the ulceration had advanced to perforation, as early as the eighth or ninth day. Stoll relates a case, where extensive sloughs were formed in the ileum as early as the seventh day,[e] and Boudet has published minute particulars of a case, fatal at the end of five and a-half days, in which deep ulcers, with partially detached sloughs, were found in the bowel.[f] There is even reason to believe, that in certain rare cases, ulceration may commence as early as the first or second day (see page 438). On the other hand, of four cases examined by Chomel, in which ulceration had not commenced, 2 died on the eighth, 1 on the eleventh, and 1 on the twelfth, day. Louis also mentions a case where ulceration had not commenced on the twelfth day. The ulceration always commences in the glands nearest to the cæcum;[g] and, in most cases, it attacks the agminated, before the solitary glands.

c. Third Stage. The stage of the pythogenic ulcer is that which intervenes between the separation of the morbid deposit, or the detachment of the sloughs, and the commencement of cicatrization. It is impossible to fix its limits, as they usually vary in the different ulcers of the same bowel. The sloughs may be found detached from the ulcers nearest to the cæcum, as early as the fourteenth or fifteenth day, but adherent to the ulcers higher up, as late as the third week, or even later. The pythogenic ulcers may be distinguished from other ulcers of the intestine by the following characters:—1. They have their seat in the lower third of the

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[c] Forget, 1841, p. 122.
[d] Louis, 1841, ii., 60.
[e] Forget, 1841, p. 116.
[f] Boudet, 1846.
[g] Chomel records one case, fatal on the tenth day, in which ulceration commenced in the patches farthest from the cæcum, but such an occurrence is quite exceptional (Chomel, 1834, obs. 4).

small intestine, and their number and size increase towards the ileo-cæcal valve. 2. They vary in diameter from a line to an inch and a half. Close to the cæcum, a number of ulcers may unite to form a mass of ulceration, several inches in extent. 3. Their form is elliptical, circular, or irregular. They are elliptical, when they correspond to an entire Peyer's patch; circular, when they correspond to a solitary gland; and irregular, when they correspond to a portion of a Peyer's patch, or when several ulcers unite to form one. 4. The elliptical ulcers are always opposite to the attachment of the mesentery. They do not form a zone encircling the gut (as may sometimes be observed in the case of the tubercular ulcer), but their long diameter corresponds to its longitudinal axis. 5. Their margin is formed by a well-defined fringe of mucous membrane, detached from the sub-mucous tissue, a line or more in width, and of a purple or slaty-grey colour: this is best seen, when the bowel is floated in water. After the separation of the sloughs, there is no thickening or induration of the edge, as in the tubercular ulcer. 6. Their base is formed by a delicate layer of submucous tissue, or by the muscular coat, or occasionally, by nothing more than peritoneum. There is no deposit of morbid tissue at the base of the ulcer, although sometimes fragments of the yellow sloughs may be seen adhering to both the base and edges.

d. Fourth Stage. The cicatrization of the pythogenic ulcer takes place in this way:—The surface of the ulcer becomes covered with a delicate shining layer of organizable lymph, which is dove-tailed, so to speak, between the muscular coat and the detached fringe of mucous membrane. The latter gradually becomes adherent to the subjacent new tissue, from the circumference towards the centre, until, at last, the healthy mucous membrane merges insensibly into the serous-looking lamina. The latter, at first, cannot, like ordinary mucous membrane, be moved upon the subjacent coat, but after a time it becomes movable, and, according to Rokitansky it becomes covered with villi. In cases where an entire Peyer's patch has sloughed out, it is almost inconceivable that the glandular tissue can be restored, but this is a point which requires investigation.

The resulting cicatrix has the following characters:—It is slightly depressed, firmer, less vascular, and smoother than the surrounding mucous membrane. When held up to the light, the bowel appears thinner at this part. The depressed spot seldom exceeds two or three lines, but may amount to half an inch, in diameter. It is never surrounded by any puckering, and it never

causes any diminution in the calibre of the gut. According to Chomel,[h] all traces of the ulcers after a short time disappear; but Barrallier[i] mentions cases where the cicatrized ulcers were distinct at the end of four or five years, and Rokitansky[k] remarks, that he has discovered cicatrices, answering to the above description, thirty years after an attack of enteric fever.

The length of time between the separation of the sloughs and the commencement of cicatrization varies; but, as a rule, the reparative process does not commence until the end of the third week of the disease. The time necessary for the cicatrization of each ulcer is, probably, about a fortnight. In one case, where the primary fever lasted three weeks, but where death occurred from complications, about the fortieth day, I found all the ulcers in the ileum cicatrized. Cicatrization commences in the ulcers nearest to the cæcum, and proceeds upwards. Consequently, when death occurs in the fourth or fifth week of the fever, the intestine may appear, at first sight, most diseased, one or two feet above the cæcum. As already stated, in cases where death occurs during a relapse, the cicatrices of the first attack may be found co-existing with the fresh deposit and recent ulcers of the relapse (see page 502).

Atonic Ulcers. The process of cicatrization is occasionally delayed, sometimes for weeks after the termination of the primary

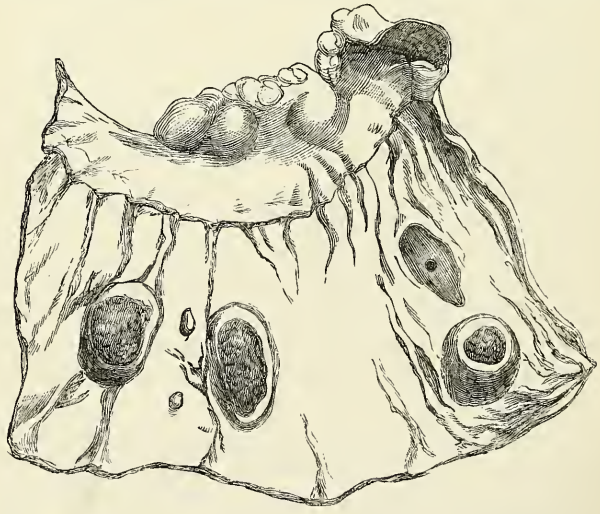

Fig. 10.—Pin-hole perforation in the ileum of a girl aged 10, who died on the 18th day of an attack of pythogenic fever. The perforation is seen in the centre of the ulcer, on the right hand side of the cut.

[h] Chomel, 1834, p. 128. [i] Barrallier, 1861, p. 105. [k] *Op. cit.* ii. 73.

fever. The ulcers pass into a chronic state, or, as some pathologists say, they become *atonic*. As a rule, all ulcers found after the fourth week of the disease, not undergoing cicatrization, may be regarded as atonic. These chronic ulcers may cause severe diarrhœa, or may lead to perforation. (See pages 507 and 508.)

Perforation. The pythogenic ulcer frequently extends through both layers of the muscular coat, leaving nothing but the peritoneum, and occasionally the peritoneum itself is perforated. Perforation may take place in three ways. 1. In the first place, it may be due to molecular disintegration, or to an extension of the ulcerative process. The opening is then always minute and rounded, just large enough to admit a pin or a stocking-wire. One or two small perforations of this sort may be seen at the base of the ulcer (fig. 10). This, according to my experience, is the most common mode. 2. A considerable portion of the peritoneum may slough, and the perforation may result from the partial or

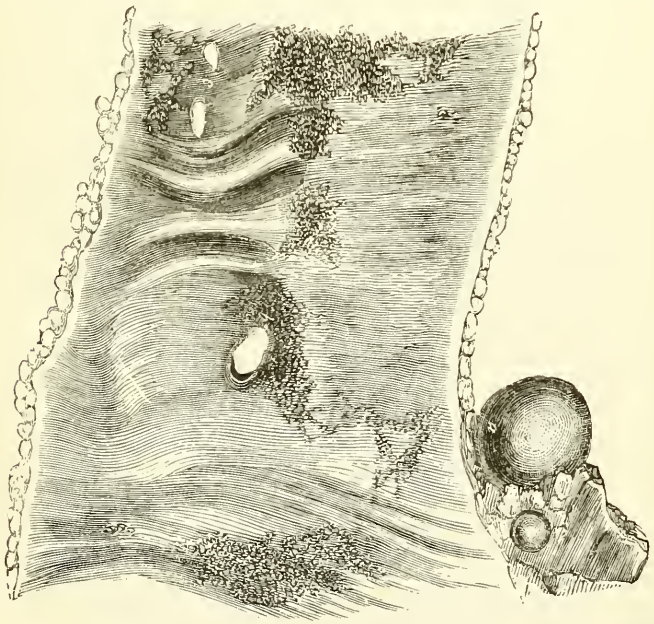

Fig. 11.—Semilunar perforation formed by the partial detachment of a slough of the peritoneum. *a*. Enlarged mesenteric gland. *b*. Dead, white portion of peritoneum, surrounded by increased vascularity: the opening is seen at lower end. *c*. Flakes of lymph. (See Case XXXV. p. 511).

complete detachment of the slough (fig. 11). In this case, the opening may be of considerable size. Dr. Lyons mentions a case, fatal not later than the fourteenth day, in which, on opening the bowel, the sloughs corresponding to several entire Peyer's patches fell out, leaving large oval apertures; in this case there was also

extensive peritonitis.[l] 3. The perforation may result from rupture of the denuded peritoneum. Some observers have doubted if this ever occurs, but the elongated linear appearance of the perforation in certain cases admits only of this explanation. Dr. Bristowe, indeed, seems to think, that the perforation is, in most cases, due to

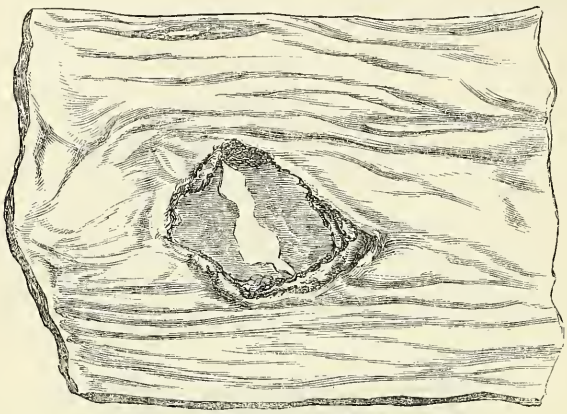

Fig. 12.—Perforation produced by rupture of the denuded peritoneum, from a case of pythogenic fever, fatal on the 25th day.

laceration.[m] This mode of perforation may possibly account for the circumstance, that perforation is so common in cases of a latent character, where the patients have not been sufficiently prostrate to confine them to the recumbent posture.

In the great majority of cases, the perforation is situated in the ileum, within 12 inches of the ileo-colic valve. Of 11 cases, of which I have notes, it was found in the lower foot of the ileum in 9; in the 2 other cases, it was situated 18 inches from the valve. Of 10 cases collected by Louis, the opening was in the lower foot of the ileum, in all. Bartlett, however, mentions one case, where it was as high as 44 inches,[n] and Bristowe found it in one instance at a distance of two yards, from the valve.[o] On the other hand, there are not wanting examples, where the perforation is found to result from an ulcer in one of the solitary glands of the colon. Chomel[p] and Brinton[q] have each recorded one case, and Forget[r] two cases, where death was due to perforation of the colon; a fifth has come under my own notice.[s] In 2 of these 5 cases, the opening was at the junction of the transverse with the descending

[l] Lyons, 1861, p. 245. [m] Bristowe, 1860, p. 113.
[n] Bartlett, 1856, p. 79. [o] Bristowe. 1860, p. 113. [p] Chomel, 1834.
[q] *Trans. Path. Soc.* ix. 199. [r] Forget, 1841, p. 354.
[s] Ibid. xiii. 65.

colon; and in the three others, at the junction of the sigmoid flexure with the rectum.

As a rule, there is only one perforation; but occasionally two, three, or even seven, have been found.

The contents of the bowel are usually prevented from escaping through the opening, in large quantities, by surrounding adhesions; and, in most cases, the peritonitis has a tendency to be circumscribed. The perforation, also, when small, is occasionally found closed up by lymph, as if undergoing a spontaneous cure. Circumscribed peritoneal abscesses occasionally result from perforation. These abscesses may induce ulceration or sloughing of the parietal peritoneum,[t] or may even open externally,[u] or into another portion of the intestinal canal; and it is probable that, in in rare cases, after the matter discharges itself in this way, recovery takes place (see page 510).

The extent of the intestinal disease, in pythogenic fever, varies greatly in different cases. The number of diseased Peyer's patches may vary from two or three to thirty or forty. At the upper part, the transition between the diseased and the healthy patches is usually rather abrupt; and proceeding downwards, after the first diseased patch, all are usually diseased. An extensive mass of disease is often found at the lower end of the ileum, terminating abruptly at the valve. There is no relation whatever between the extent of the intestinal disease, and the severity of the cerebral or abdominal symptoms (see pages 480 and 497).

The intestinal lesions are the same in children as in adults. Extensive deposit, however, in the submucous tissue, followed by sloughing of the diseased patches in large masses, is less common. The solitary glands, also, have a greater tendency to be attacked, and extensive ulceration, or perforation, is comparatively rare (see page 511).

The morbid appearances presented by the agminated and solitary glands of the ileum, above described, are constant in, and peculiar to, pythogenic fever. They characterize neither typhus, nor any other disease. '*Il faut,*' says Louis, '*non seulement la considérer comme propre à l'affection typhoïde, mais, comme en formant le caractère anatomique, ainsi que les tubercules forment celui de la phthisie.*'[x] Care, however, must be taken not to

[t] JENNER, 1853, p. 286. [u] *Brit. Med. Journ.* 1861, i. 602.
[x] LOUIS, 1841, i. 199.

set down every unusual appearance of the parts in question to pythogenic fever. 1. In young children, Peyer's patches are naturally more distinct than in adults; but this condition bears no resemblance to that resulting from pythogenic fever. 2. The appearance likened by French pathologists to a shaven beard, is well known not to be characteristic of pythogenic fever, but to be met with after death from many diseases, and even to be compatible with perfect health. This circumstance was pointed out nearly thirty years ago by Chomel,[y] and has been insisted on by many subsequent observers.[z] The appearance in question is independent of any morbid deposit, but is probably due to the action of the intestinal gases on the blood contained in the capillaries, in the folds of the membrane lying between the glandules. 3. The agminated and solitary glands of the ileum may be the seat of tubercular deposit and ulceration; but no experienced pathologist could mistake the cup-shaped tubercular ulcer, with its edges and base indurated from deposit of tubercle, for the pythogenic ulcer already described. Moreover, intestinal tubercle is probably always accompanied by tubercle elsewhere. 4. The appearances, which are most likely to be mistaken for the lesions of pythogenic fever, are those which are occasionally met with after death from cholera, variola, scarlatina, erysipelas, and pyæmia. In these diseases, the solitary and agminated glands are occasionally found slightly thickened and elevated. The enlargement, however, is always slight; it does not pass through the successive stages observed in the lesions of pythogenic fever; it very rarely produces ulceration;[a] and it is not accompanied by enlargement of the mesenteric glands.[b] These are not the lesions of pythogenic fever in an early stage, for they are not found any more advanced, when death does not occur until the twentieth or thirtieth day of the illness. Moreover, they are only present in exceptional cases of the diseases in question; whereas, the lesions above described are never absent in pythogenic fever.

5. *The large Intestines.* The colon is, in most cases, more or less distended with gas. Its distension is sometimes so great, that it forms numerous coils, which obscure and displace the other

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[y] Chomel, 1834, p. 149.

[z] Jenner, 1853, p. 287; Jacquot, 1858, p. 252; Barrallier, 1861.

[a] Dr. Anderson records a case of scarlatina, and a case of variola, where Peyer's patches were found ulcerated. Such occurrences are extremely rare, and the small shallow ulcer thus produced, is very different from the ulcers of enteric fever. At the same time, the possibility of enteric fever co-existing with some of the diseases mentioned is to be borne in mind. This, in fact, is the light in which Dr. Anderson was disposed to view his cases. (Anderson, 1861 p. 115).

[b] Rokitansky, *Path. Anat.* Syd. Soc. Transl. ii. 89.

viscera. Louis mentions a case, where the liver was displaced in this way so high, that the hepatic dulness was mistaken during life for pneumonia. The mucous membrane of the colon presents the same varieties, as to colour and consistence, as were observed in the small intestine. These abnormal appearances are common in many other diseases, besides pythogenic fever.

The solitary glands of the colon may become the seat of morbid deposit and ulceration, like those of the ileum. Ulcers of the large intestines were observed in 14 out of 46 cases by Louis, and in 7 out of 20 cases by Jenner. As a rule, the disease is confined to the cæcum and ascending colon, but it may reach as far as the sigmoid flexure. The ulcers are usually small and round, but they sometimes extend, so as to measure fully an inch and a-half in length, and then their large diameter is usually transverse, corresponding to the folds of the gut.

The lesions of dysentery occasionally coexist with those of pythogenic fever (see page 507).

6. *The Mesenteric Glands* are invariably enlarged,[c] but their appearance varies considerably, according to the stage of the disease at which death occurs. They begin to enlarge at the very commencement of the disease (see pages 438-9), and go on increasing in size, contemporaneously with the enlargement of the intestinal glands, until about the tenth or fourteenth day of the fever. At this time, they are sometimes found equalling or exceeding a pigeon's egg in size, their consistence is tolerably firm, or slightly softened, and their colour is rosy-red or purplish. As soon as the morbid material begins to be detached from the intestinal glands, the mesenteric glands usually decrease in size, and become softer; but they are found to be considerably larger than natural, as late as the thirtieth day, or later. When death does not occur until after the sixth week, they are often unusually small, shrivelled, tough, and either very pale, or of a grey or bluish colour. In cases, however, where death is due to a relapse of the primary fever, many of them may be found greatly enlarged, as late as eight or twelve weeks from the first commencement of the illness (see pages 502 and 548).

When a section of one of the glands is examined about the twelfth or fourteenth day, several small circumscribed masses of opaque, pale-yellow, friable deposit may occasionally be observed. This deposit exactly resembles that found in the intestinal glands.

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[c] Hence the designation of 'Febris mesenterica,' applied to the disease by many writers. (See pp. 395, 387, 389).

After a time, these masses become softened at their edges into a fluid resembling pus, and then, on cutting across the gland, a number of little drops of puriform fluid may be seen, each with a central yellow slough. In rare cases, an entire gland may become converted into a collection of puriform matter, as large as a walnut, in the centre of which are detached sloughs of considerable size. These pseudo-abscesses are usually formed in one of the glands at the termination of the ileum, and they may be found as late as the sixth or eighth week of the disease. Now and then, they may be seen with nothing more than a thin layer of peritoneum separating their contents from the abdominal cavity, and occasionally they burst through the peritoneum and excite general peritonitis[d] (see page 507). Blood is sometimes extravasated from the interior of the ruptured gland into the peritoneal sac.

The morbid changes now described are usually most marked in the glands corresponding to the most diseased portions of the bowel, or to the lower end of the ileum. From this situation, they gradually diminish in size, as we proceed upwards. The meso-colic glands are also enlarged in cases where the mucous membrane of the colon is diseased. At the same time, the enlargement of the mesenteric glands is not merely the result of the irritation from the disease in the bowel, but it must be viewed as a primary anatomical lesion, like that of the intestines. In 10 of Louis's 46 cases, there were diseased mesenteric glands corresponding to perfectly healthy portions of intestine; while, in another patient, who died on the eighth day of the disease, the meso-colic glands were enlarged and softened, although the mucous membrane of the colon was perfectly healthy. Moreover, as above stated, it is probable that the mesenteric glands become enlarged as soon as those in the intestine.

The glands in the fissure of the liver, the gastric, œsophageal, lumbar, or inguinal glands, are occasionally found enlarged; but, in most cases, the enlargement is due to irritation from ulcers in the stomach or œsophagus, from erysipelas, or from blistered surfaces on the legs.[e]

7. *The Spleen* is almost invariably found to be hypertrophied when death occurs before the thirtieth day. Of 16 cases, in which death occurred before that date, I found the spleen enlarged in all: its normal weight being $4\frac{1}{2}$ ounces, the average of the 16 cases was $11\frac{2}{5}$ ounces; the smallest weighed $6\frac{1}{2}$ ounces, and the largest,

[d] See LOUIS, 1841, i. 240; JENNER, 1849 (2); ROKITANSKY, *Path. Anat.* Syd. Soc. Transl. ii. 78.

[e] See LOUIS, 1841, i. 254.

17 ounces. On the other hand, in 3 cases where death did not take place until the fifth or sixth week, the average weight of the spleen was less than 5 ounces. In 11 cases, where death occurred before the thirty-fifth day, Jenner found the average weight of the spleen to be 10 oz. 3 dr. avoird.; the smallest was 6 ounces, and the largest 14 ounces. Louis found the spleen enlarged, in at least 36 out of 46 cases, and in 17 it was three, four, or five times its normal size: most of the cases, where it was not enlarged, were fatal after the thirtieth day. The enlargement is usually greatest in persons under thirty years of age. This difference, according to age, may account for the circumstance that, the organ is, on the whole, larger in enteric fever than in typhus.

The consistence of the spleen is usually reduced. Louis found the spleen softened in 34 out of 46 cases; and in 7 it was reduced to a mass of 'putrilage.' The spleen was decidedly softened in 4 out of 14 cases examined by Jenner, and in 10 out of 21 cases dissected by myself. The softening is always greatest when death occurs before the thirtieth day. According to Rokitansky, the enlarged softened spleen is liable to spontaneous rupture.

The colour is usually darker than natural.

Masses of opaque yellowish-white deposit are occasionally met with in the spleen. Three cases have been already referred to, where these deposits became softened and burst, producing fatal peritonitis (see page 507). These deposits are not alluded to by Louis, Chomel, or Forget, and I have never met with them myself. They have been thought to consist of similar material to what is deposited in the intestinal glands; but their specific nature may be doubted. Similar deposits are often met with in other diseases, and even in typhus and relapsing fever, which appear to have an embolic origin.[f]

8. *The Liver and Gall-bladder.*—The liver is occasionally hyperæmic: it was so in 8 out of 46 cases examined by Louis; in 2 out of 15 dissected by Jenner, and in 3 out of 12 cases noted by myself; but in most cases, its colour is normal, or it is unusually pale. The organ was softer than natural in 32 out of 73 cases examined by Louis, Jenner, and myself. This softening is perhaps occasionally cadaveric; but, in one case, I have found the quantity of oil in the secreting cells much increased (see page 235).

[f] Waters records a case of typhus, in which the spleen contained friable yellow deposits; there was no disease in the bowel (Waters, 1847). Another case has been already referred to (see page 197). The possibility of the so-called 'typhous deposits,' in the spleen being due to embolism, deserves investigation. (See also p. 507).

Frerichs has met with cases, in which the liver was in a state of acute atrophy, and he remarks that it not unfrequently contains leucine, tyrosine, and other products of disintegrated tissue.[g] Louis and Frerichs each record a case where the liver contained a number of circumscribed masses of morbid deposit, the exact nature of which was doubtful (pyæmic ?)[h]

The lining membrane of the gall-bladder is very liable to become inflamed in pythogenic fever, without producing any symptoms during life. Illustrative cases have been recorded by Andral,[i] Louis,[k] Budd,[l] Rokitansky,[m] Frerichs,[n] etc. The inflammation assumes different forms. Sometimes it is of a catarrhal nature, and the gall-bladder is found to contain pus, as in three cases recorded by Louis. At other times, according to Rokitansky it takes a diphtheritic character, and the gall-bladder and biliary passages are found lined with tubular investments of exudation, which may block up the passages, and cause dilatation of the smaller ducts. Thirdly, it may take the form of ulceration : cases where the mucous membrane has been found ulcerated, have been recorded by Andral, Jenner,[o] and Trousseau ;[p] and two instances have been already referred to, where the ulceration ended in perforation and fatal peritonitis (see page 507). There is no evidence that these morbid conditions of the gall-bladder are due to any specific morbid deposit, like the deposit in the intestinal glands. As Frerichs has shown, they are met with in other febrile diseases, besides enteric fever.

In a large proportion of cases, where the disease has lasted three or four weeks, the bile is thin, watery, and almost colourless, although the cystic duct is pervious. This condition has been noticed by many observers ; but I have rarely, if ever, met with it in typhus. According to Martin Solon, the contents of the gall-bladder, when they present the appearance now described, are of acid reaction.[q]

9. *The Pancreas* is usually healthy, but occasionally it is of a rosy or livid hue from hyperæmia, or its consistence is reduced.

10. *Peritoneum.* From the remarks already made, it is obvious, that recent peritonitis is far from being an uncommon lesion in pythogenic fever. Its various causes have been already considered (see pages 507-8).

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[g] *Dis. of Liver*, Syd. Soc. Transl. i. 215.

[h] Louis, 1841, i. 118 ; Frerichs, *Op. Cit.* i. 172.

[i] *Clin. Méd.* 4me. ed. ii. 549. [k] Louis, 1841, i. 281.

[l] *Dis. of Liver*, 3rd ed. p. 195. [m] *Path. Anat.* Syd. Soc. Transl. ii. 160.

[n] *Op. cit.* ii. 454. [o] Jenner, 1849 (2). [p] Trousseau, 1861, p. 203,

[q] Solon, 1847.

d. Organs of Circulation and Blood.

1. *The Pericardium* is usually healthy. Occasionally, it contains a few drachms of serous fluid; but out of 84 cases noted by Louis, Jenner, and myself, in only one (Louis) was the fluid of a sanguinolent hue; and in only one (Jenner) did it contain shreds of lymph, indicative of recent pericarditis (see page 236).

2. *The Heart.* Softening of the muscular tissue of the heart is less common than in typhus. Rokitansky observes, that, although it may be flabby and pale, it is free from 'that softening of its 'substance, described by Stokes as occurring in the typhus fevers 'of Ireland.' Softening of the heart, however, like that observed in typhus (see page 236), is more common than might be inferred from this statement. Louis found marked softening, sometimes associated with thinning of the walls, in 15 out of 47 cases; and he showed that this condition was independent of the external temperature, or of the length of interval between death and the autopsy. Jenner found the heart soft and flabby, or flabby only, in 6 out 11 cases; and Chomel noted marked softening in 7 out of 30 cases. As in typhus, the softening may extend over the whole heart, or it may be limited to the left ventricle.

3. *The Endocardium*, in cases where the blood is fluid, occasionally presents a dusky-red discolouration. This appearance, however, is much rarer than in typhus. Jenner noted it in only 3 of 16 cases, and in all it was slight (see page 237).

4. *The Blood.* A dark, liquid condition of the blood is rarer than in typhus, and firm white coagula are more common. Louis found white coagula in the heart in more than one half of his cases. Out of 14 cases, Jenner found the blood fluid in 3, and coagulated into pale fibrinous clots, in 10. Of 9 cases noted by myself, the blood in the right side of the heart contained pale fibrinous clots in 6; in 1, it resembled currant jelly; and in 2, it was dark and fluid. On the other hand, Chomel found the blood black and fluid in 15 out of 30 cases, and containing fibrinous clots, in only 6. There is a close relation between the condition of the blood and the symptoms during life. When death has been preceded for some days by the typhoid state (see page 497), the blood is usually dark and fluid; in other cases, as, for example, when death is due to perforation or pneumonia, it often contains fibrinous coagula. The fluid condition of the blood is probably due to the same cause as in typhus (see page 238).

Lehmann states, that during the first week of enteric fever, the blood resembles that of plethora, the corpuscles and solids of the serum, especially the albumen, being increased, but that from

about the ninth day, the corpuscles and the solids of the serum diminish, with a rapidity proportionate to the intensity of the intestinal affection.[r] Virchow maintains that the number of white corpuscles is always increased, while the fibrine is diminished. The increase of the white corpuscles he attributes to the enlargement of the mesenteric and Peyerian glands.[s] Virchow and other observers have also discovered in the blood of enteric fever, and of other typhoid diseases, minute reddish-black bodies, smaller than red corpuscles, which they regard as red corpuscles undergoing disintegration.[t] When the blood is dark and fluid, the red corpuscles are often crenate and mis-shapen, as in typhus.

e. Organs of Respiration.

1. *The Epiglottis* was found by Louis to present signs of recent inflammation in 10 out of 46 cases. It was œdematous, congested, ulcerated, or covered with false membrane. In all of the cases, death occurred at an advanced stage of the disease.

2. *Larynx and Trachea.* The various forms of inflammation to which the larynx is liable, in the course of pythogenic fever, have been already alluded to (page 503). It is only necessary now to add a few words concerning that form, in which the mucous membrane is found to be ulcerated. This lesion is far from being common. Louis met with it in only 3 of 96 cases; Chomel, in 1 of 42 cases; and Jenner, in 1 of 15 cases, examined after death. Rokitansky observes that 'secondary pharyngeal typhus occurs 'much more rarely than secondary laryngeal typhus,'[u] but Louis and Jenner found ulcers in the pharynx and œsophagus to be far more common than ulcers in the larynx (see page 536). The ulcers in the larynx are usually situated near the posterior junction of the vocal cords. They are sometimes superficial; at other times, they spread by sloughing, and are so deep as to lay bare the subjacent cartilages, or produce perforation of the entire larynx, with escape of air into the cellular tissue (see page 504). They are rarely, if ever, found before the fifteenth day of the disease. Like the ulcers in the pharynx and œsophagus (see page 537), there is no evidence that they are due to the sloughing out of a morbid material ('typhous matter') deposited in the sub-mucous tissue. As far as I know, no such deposit has ever been found in the larynx, either co-existing with ulcers, or independent of them.

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[r] *Physiol. Chem.* Day's Transl. ii. 266.
[s] *Cellular Path.* Chance's Transl. p. 167. [t] Ibid. p. 225.
[u] *Op. cit.* ii. 79.

When we remember the remarkable tendency to ulceration exhibited by the pharynx, the œsophagus, the stomach, and the gall-bladder, in the advanced stages of enteric fever, it is not surprising that inflammation of the larynx should occasionally lead to the same result. Trousseau says that laryngeal ulcers are most common in persons who have been kept on too low diet, and he quotes the experiments of Chossat, to the effect that the production of ulceration is one of the effects of inanition. Moreover, ulceration of the larynx is occasionally found in typhus (p. 239). Trousseau justly observes: 'Ces lésions s'expliquent sans qu'il 'soit besoin d'invoquer une localisation spéciale de la maladie, 'analogue à celle qui se fait du côté de l'intestin.'[x]

3. *The Bronchi* are often filled with frothy mucus, while their lining membrane is much congested; but these appearances are, on the whole, less common than in typhus (see page 239).

4. *The Lungs* are occasionally found healthy, especially when death takes place suddenly by peritonitis; but in most cases, they exhibit one or other of the morbid conditions, described under the head of typhus.

Hypostatic consolidation is, on the whole, less common than in typhus. Jenner did not observe it in any of 15 cases. I have noted this condition, however, in 7 out of 19 cases; and in 4 of the 7 cases, the consolidation was so great that the most dependent portions of the lungs sank in water. In all of the 7 cases, the typhoid state had been well-marked prior to death. Louis also noted hypostatic condensation in 19 out of 46 cases. He applied to it the designations 'splénisation ou carnification,' (terms which are now given to two entirely different lesions); but he accurately described its characters, and its points of distinction from true pneumonia. Thus, it was limited to the most dependent portions of the lungs; its cut surface was non-granular, and discharged, when squeezed, a quantity of reddish serum, without any bubbles of air; and the condensed tissue not only sank in water, but was more tenacious than in the natural state (see pages 183 and 239).

Œdema of the lungs is occasionally met with, and, according to my observations, is most common in the upper lobes (see page 239).

True pneumonia is much more common than in typhus. It existed in 8 out of 19 cases noted by myself; in 17 out of 46 cases examined by Louis, and in 12 out of 15 cases dissected by Jenner. It is usually lobular. In rare cases, the circumscribed condensed

[x] Trousseau, 1861, p. 203.

patches appear to become converted into small abscesses, or they pass into gangrene. In one of Louis' cases, a number of small abscesses were found scattered through the lung, and one similar case has come under my notice. In two cases, I have found a patch of gangrene in the lung, as large as a walnut.

In two cases of enteric fever, I have found in the lungs the appearance described as *vesicular pneumonia*, in which the exudation appeared to be thrown out into the air-cells, the condensed tissue being studded with a number of small white masses resembling miliary tubercules. I have never met with any appearance to justify the appellation of *typhoid pneumonia*. It has been stated that the exudation thrown out into the lungs, sometimes presents the same appearance under the microscope, as the matter deposited in the intestines, but all observations of this sort must be viewed with considerable caution, inasmuch as the intestinal deposits have no peculiarity of structure, by which they can be recognized.

Recent tubercle is occasionally met with in the lungs, in protracted cases of enteric fever (see page 503).

5. *The Pleuræ* exhibit signs of recent inflammation oftener than in typhus. Recent adhesions or effusion of lymph existed in 6 out of 19 cases examined by myself, and in 6 of 15 cases noted by Jenner, but only in 2 of 46 cases recorded by Louis. In 19 out of 46 cases, Louis found a greater or less amount of reddish serous effusion in the pleuræ; in most of these cases there was hypostatic consolidation of the lungs (see page 503).

6. The *Bronchial Glands* are occasionally enlarged when the lungs are inflamed, as is often the case in ordinary pneumonia.

f. Nervous System.

1. *The Cerebral Membranes* are less frequently congested than in typhus. Jenner found the dura mater normal in every one of 15 cases; the pia mater was congested in 5 cases, but in 4 of the 5 cases, the congestion was confined to the larger vessels. Louis found increased vascularity in almost one-half of 46 cases; but in only 11, was the congestion considerable. There is no relation between the severity of the cerebral symptoms during life, and the vascularity of the cerebral membranes found after death. The same remarks are applicable here as in typhus (page 240).

I have never met with hæmorrhage into the cavity of the arachnoid in pythogenic fever. Louis makes no mention of it, and it did not exist in any of 19 cases examined by Jenner. Chomel, however, appears to have observed it in one case [y] (see page 241).

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[y] Chomel, 1834, Case 18.

It is not often that the membranes can be torn from the brain with increased facility. Jenner noted this condition in only 1 of 9 cases (see page 241).

2. *Intra-Cranial Fluid.* Effusion of serous fluid at the base of the brain, into the lateral ventricles, and beneath the arachnoid, is almost as common as in typhus, although the quantity, is, on the whole, less. Louis and Jenner met with more or less sub-arachnoid serosity in 37 out of 61 cases; but in only 5 of the cases, was it considerable in amount. The fluid thrown out into the several localities mentioned, is always *colourless and transparent*, and is no more a sign of inflammation, than it is in typhus (page 242.) Two exceptions to this rule are recorded by Louis (*Obs.* 17 and 25): in one, the fluid in the arachnoid was turbid and contained a few albuminous flakes, but the vascularity of the membranes was not increased; in the other, a recent false membrane was found on the visceral surface of the dura mater; but here there was pyæmia, and the symptoms of meningitis did not supervene, until after convalescence had commenced (see also pages 505-6).

4. *The Cerebrum* and *Cerebellum* are usually normal. Increased vascularity of the cortical substance (in 17 of 46 cases, Louis), or of the medullary portion (in 9 of 61 cases, Louis and Jenner), is occasionally met with. There was no trace of softening or induration of the cerebral substance in any of 15 cases examined by Jenner. Louis found induration of the brain in 7, and softening in 7 out of 46 cases; but these appearances were mostly uniform over the entire brain, and no relation could be traced between them and the severity of the cerebral symptoms during life. The remarks (page 244), made under the head of typhus, are also applicable here.

It was clearly shown by Louis and Chomel, that the morbid appearances found in the brain and its membranes in enteric fever, were equally common after death from other acute diseases, especially pneumonia, and that no relation existed between them and the intensity of the cerebral symptoms. My own observations agree entirely with their statements (see pages 243 and 497).

5. *The Spinal Cord* was examined by Louis in 6 cases, but presented nothing abnormal.

6. *The Sympathetic Ganglia*, according to Virchow, often contain an unusual amount of pigment in the interior of the ganglion-cells.[z]

[z] *Cellular Path.* Eng. ed. p. 257.

g. Urinary Organs.

1. *The Kidneys* are often found congested, when death occurs before the end of the third week. In two instances, I have found the hyperæmia so intense, that the organs presented a dark chocolate colour. When death occurs at a later stage, the kidneys are occasionally pale and slightly increased in size. Occasionally, the uriniferous tubes are found gorged with epithelium; but this condition, as well as that of enlargement, is rarer than in typhus. Louis found the kidneys hyperæmic in 17, and unusually pale in 5, out of 42 cases; the former appearance was most common in cases fatal between the eighth and the fifteenth day; in all the cases where the kidneys were pale, the duration of the illness had been considerably longer.

2. *The Bladder.* The mucous membrane of the bladder is occasionally congested. In one instance, Louis found a minute ulcer near the opening of the urethra. Diphtheritic inflammation of the bladder is occasionally met with in enteric fever, and in other acute specific diseases. The mucous membrane is congested and coated with patches of fibrinous exudation, beneath which the surface is abraded.[a]

Nature of the Deposit in the Intestinal and Mesenteric Glands.

While many French, and some English, writers have regarded the intestinal affection as an exanthem, and compared it to the eruption of variola, the pathologists of the Vienna School have taught for the last twenty years, that the morbid deposit (*Typhusmasse*) occurring in the intestinal glands and elsewhere, is an albuminous exudation, which, like tubercle or cancer, depends on a morbid condition of the blood, and undergoes a peculiar development. A specific 'typhous cell' has been described and figured by Gruby, Vogel, J. H. Bennett, and others. It is maintained, that the deposition of this 'typhous matter' is not restricted to the intestinal and mesenteric glands, but that it is met with in the spleen, gall-bladder, stomach, œsophagus, larynx, lungs, etc. As already stated, however, there is no evidence that the lesions found in these organs are due to the deposit of any material, resembling that found in the intestinal glands.

Again, the matter found in the intestinal and mesenteric glands has no specific structure, by which it can be distinguished from other exudations. Its microscopic characters vary greatly in

[a] Rokitansky, *Op. cit.* ii. 227; Jenner, 1849 (2).

different cases. On examination with a quarter of an inch lens, it is sometimes found to contain cells such as are figured in the annexed wood-cut. These cells present very different appearances. They vary in diameter, from $\frac{1}{1200}$ to $\frac{1}{4500}$ of an English inch. Some are round or oval, and contain one, two, or many rounded globules, resembling nuclei. Others have an irregular outline, and contain only a few granules, or are loaded with oil. These cells

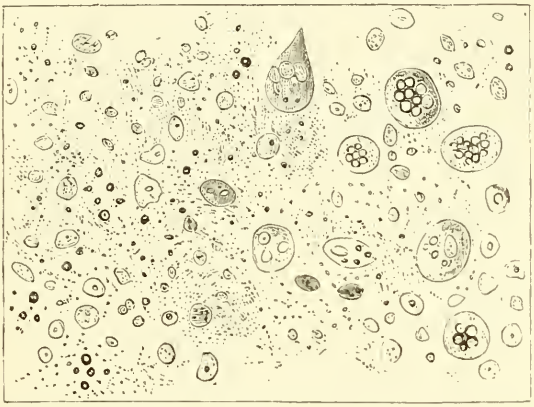

Fig. 13.—Morbid deposit from one of the solitary glands in the ileum in a case of pythogenic fever, magnified 400 diameters.

are mixed with a large quantity of granular matter and oil-globules. In many, if not most, cases, the deposit contains nothing like a nucleated cell, but only a number of small irregular corpuscles which more resemble tubercle than anything else; and sometimes even these are absent, and the substance is made up of molecules and granules. Wedl,[b] Virchow,[c] and many recent observers, insist that there is nothing specific or characteristic in the structure of the so-called 'typhous matter.'

It is remarkable, that the essential seats of disease, in enteric fever, probably all belong to the same physiological system. Kölliker has pointed out the close resemblance in structure, which a Peyer's patch bears to a lymphatic gland, and he states that the period of greatest activity of the Peyerian vesicles corresponds to that of intestinal absorption.[d] Brücke has succeeded in injecting the glandules from the lacteals.[e] Virchow also insists, that the solitary and agminated glands have nothing in common with the

[b] *Path. Histology*, Syd. Soc. Transl. p. 334.
[c] *Cellular Path.* Eng. Transl. p. 397.
[d] *Man. of Hum. Hist.* Syd. Soc. Transl. ii. 106.
[e] CARPENTER's *Princip. of Hum. Phys.* 5th ed. p. 119.

glands which pour their secretion into the intestinal canal, but that a Peyer's patch is nothing more than a lymphatic gland, spread out in single layer in the coats of the intestine.[f] Moreover, it is now generally admitted that the spleen, which is so often enlarged in enteric fever, in common with other fevers, is intimately related in its functions to the lymphatic system, both being concerned in the process of sanguification.

The circumstance, that all the organs essentially diseased in pythogenic fever belong to the same physiological system, is, in itself, an argument against the view that the material deposited has a specific character, like tubercle or cancer. Indeed, when we study the various steps of the disease in the intestinal glands, (see page 543), it may be doubted if the so-called 'typhous matter' be an exudation at all, in the strict sense of the term. It is far more probable, as suggested by Virchow, that it is always a directly continuous development of the pre-existing cellular elements of the diseased glands.

The morbid anatomy of pythogenic fever may be summed up as follows :—

1. The agminated or solitary glands of the ileum, and the mesenteric glands, are invariably diseased.

2. Many other secondary lesions are found, which are not constant or essential. The chief of these are enlargement of the spleen, ulcerations of various mucous surfaces, pneumonia, bronchitis and hypostatic consolidation of the lungs, softening of the heart, and a large amount of intra-cranial fluid.

3. There is no specific 'typhous exudation,' and no evidence that the secondary lesions above mentioned are due to the deposit of a material, like that found in the intestinal and mesenteric glands.

4. There are no signs of inflammation in the brain or its membranes, to account for the cerebral symptoms.

Section XIII.—Treatment.

a. Prophylactic Treatment.

As in typhus, the subject of prophylaxis involves the consideration of the measures calculated to prevent the origin, as well as the propagation, of the pythogenic poison.

1. *Measures for Preventing the Generation of the Pythogenic Poison.*

These measures are obvious, from the remarks already made

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[f] *Op. cit.* p. 192.

under the head of etiology. *Instead of cutting off thousands annually, enteric fever would be a rare disease, if we could prevent the products of fæcal fermentation from entering our houses and polluting our drinking-water.* It is the duty of every householder to see that the house-drains and sinks are properly trapped. Even if the trapping be good, gases will escape from drains, if the supply of water be deficient. In all water-closets, care should be taken that the water rises in the pan above the level of the lower opening, and if the cistern does not supply a sufficient quantity, it must be obtained from other sources. When bad smells escape from sinks or drains, disinfectants ought to be used, but they must never be substituted for measures calculated to prevent the escape of the products of fæcal fermentation into the interior of houses. *It must not be forgotten, that the poison of enteric fever, although often accompanied by bad smells, may be itself inodorous* (see page 454).

The best disinfectants are chloride of lime and Condy's manganic fluids. Chloride of lime may be used in the proportion of a pound to a gallon of water. Condy's red fluid may be diluted with fifty times its bulk of water. The solutions of chloride of zinc, and of perchloride of iron, are also good disinfectants.

Obstructions of house-drains, and leakages from drains and cess-pools, ought to be rectified without delay. When the drains or cess-pools of a house require to be taken up for the purpose of repair or cleansing, disinfectants ought to be used, and thorough ventilation enforced. It is also advisable, when possible, that the residents should absent themselves while the operations are going on. *Enteric fever has often broken out, in consequence of the measures resorted to for its prevention.*

No water ought to be drunk, which is contaminated by leakage, or filtration from sewers, cess-pools, or foul ditches. When possible, the water supplied in towns, or derived from surface-wells, ought always to be filtered before use.

The above measures, and many others which will suggest themselves according to circumstances,[g] require especial attention during the autumn, and in the case of persons below the age of thirty, who have only recently been exposed to the nuisances mentioned.

2. *Measures for Preventing the Propagation of Pythogenic Fever.* As it is probable in those cases where pythogenic fever is propagated from the sick to persons in health, that the alvine evacuations constitute the chief medium of communication, they ought

[g] The reader is referred to an excellent *Memorandum* on this subject, issued by the Privy Council.

always to be disinfected by one of the preparations already mentioned, before they are emptied into privies, drains, or cess-pools; and care should be taken that they are never thrown on places, where they can soak into the sources of drinking-water. The bedding, and all articles of clothing, soiled with the discharges of the sick, ought to be disinfected, and, at the same time, thorough ventilation should be enforced in every apartment of the infected dwelling.

But, in addition to these precautions, attention must at once be directed to the *original cause* of the fever, which, in most cases, will be discovered, and *the persistence of which is, in my opinion, a far more fertile cause of fresh cases in a house, than a poison derived in any way from the person first infected.* Such nuisances as have been mentioned under the head of etiology, are to be sought for and remedied; and until this is done, it is advisable, when practicable, that all the inmates below thirty years of age should absent themselves from the infected house.

b. Remedial Treatment.

1. *Hygienic.*

The rules laid down for the hygienic treatment of typhus (see page 251), are also applicable to pythogenic fever, with this addition, that, in consideration of the intestinal lesions, especial care is required with regard to *diet.* Toast-water, barley-water, rice-water, a thin solution of gum arabic and apple-water may be given as drinks. Although one of the main objects in treatment is to nourish the patient from the commencement of the attack, we must guard against giving food *too often,* or of such a quality as to irritate the bowels (see page 253). All food should be given in a fluid form, and fruit of every sort is to be strictly prohibited. Milk, beef-tea, custards, and arrowroot, are the main articles on which we ought to rely. Beef-tea, thickened with arrowroot, is an excellent form of nourishment in enteric fever. The 'Preserved Meat Juice,' manufactured by Messrs. Gillon, of Leith, and sold in small canisters, is a preparation which is of great service in the treatment of fevers. It differs from ordinary beef-tea, in consisting principally of osmazome, with the salts and sapid principles of meat; and, as appears from Dr. Christison's observations, it not only acts as a nutrient, but *it diminishes the waste of the tissues.*[h]

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[h] See *Edin. Month. Journ.* Jan. 1855. A tin of four ounces, sold for sixpence, makes sixteen ounces of strong beef-tea. In the ordinary way, this quantity requires a pound of the finest beef, which costs ninepence or tenpence.

2. *Therapeutic Treatment.*

Numerous methods have been recommended for the treatment of enteric fever, a few of which may be alluded to, before proceeding to the consideration of what, according to our present knowledge, appears to be the rational method.

Blood-letting. In studying the history of continued fevers, it is found that blood-letting has been far more commonly practised in enteric fever, than in typhus. Early in the present century, this practice received a fresh stimulus, from the promulgation of the doctrines of Broussais, who, believing the fever to be only a symptom of local inflammation, recommended blood-letting at every stage of the disease. Louis, Chomel, and many other writers, although disputing the doctrines of Broussais, practised blood-letting in the early stage; while Bouillaud's treatment consisted in frequent copious abstractions of blood from a large vein, with leeches and cupping in the intervals. For example, one of Bouillaud's patients, suffering from enteric fever complicated with pneumonia, was bled largely six times from the arm, was cupped three times, and had 60 leeches applied to the chest and abdomen. Under this treatment ('émissions sanguines coup sur coup'), it was alleged that the mortality was much smaller than in the practice of Louis and Chomel, who bled more sparingly.[1] Although, in the discussion, which followed the enunciation of this practice, it was shown that Bouillaud's success was founded on fallacious statistics, his treatment is still pursued in many parts of the continent, where, from time to time, we hear of valuable lives being sacrificed to it.

Bouillaud's practice never obtained favour in this country; but there are many practitioners who think, that much advantage is derived from the application of leeches, for the relief of the abdominal symptoms. In several cases, at an early stage of the disease, I have applied leeches for this purpose, both to the abdomen and round the anus, and I have usually found their employment followed by marked relief to the pain, and sometimes by subsidence of the diarrhœa. At the same time, I have often observed equal relief afforded by the constant application of poultices and warm fomentations, so much so, that I am inclined to think that the good effects of leeches are, in a great measure, due to the fomentations which succeed them.

The Cold Affusion. Currie practised the cold affusion in all forms of continued fever; but he looked upon severe diarrhœa as

[1] Bouillaud, 1836, p. 380.

a contra-indication.[k] In 1846, Mr. Stallard, of Leicester, treated a number of cases of 'Enteric Fever,' by packing in a wet sheet. The patient was enveloped in a cold wet sheet, and covered with a blanket. After ten or fifteen minutes, he was transferred to a blanket heated before the fire, and he was covered with bed-clothes. He soon began to perspire, and sank into an undisturbed sleep, from which he awoke free from headache and pain, and greatly refreshed. One effect of the treatment was to *confine* the bowels.[l] More recently, Trousseau has recommended the cold affusions, as practised by Currie, in those cases of enteric fever which are characterized by restlessness, delirium, stupor, and other cerebral symptoms.[m] The effects of the cold affusion in enteric fever, as well as in typhus, deserve investigation (see page 259).

Quinine, in large doses, has been found equally inefficacious in enteric fever. as in typhus (see page 261). At the same time, in cases where the disease has presented a remittent character, I have frequently observed great abatement of the febrile symptoms follow its administration, in doses of one or two grains frequently repeated. M. Mazada, who employed it extensively, observed similar good effects when the fever was remittent, but found it often injurious when the fever was continued.[n] Barthez and Rilliet also report favourably on the effects of quinine in the enteric fever of children, which is so frequently remittent, its beneficial action manifesting itself in a reduction of the pulse and temperature.[o]

Emetics have been recommended in the treatment of enteric fever from time immemorial, and are still favourite remedies with many practitioners. Dr. James Jackson, of America, by a comparison of a large number of cases treated with and without emetics, has endeavoured to show that they reduce the duration, as well as the severity and rate of mortality of the disease. His remarks are well worthy of perusal.[p] There is no proof, however, that emetics ever cut short an attack of enteric fever. The cases in which they have been supposed to do so, are open to the same objection as in typhus (see page 263). At the same time, when administered within the first ten days of the disease, they often relieve the headache and gastric disturbance. Indeed, they constitute one of the best remedies for vomiting in the early stage. They ought never to be given after the twelfth day; for, when the

[k] Currie, 1797, i. 20.
[l] *Brit. & For. Med. Rev.* Jan. 1847, p. 269.
[m] Trousseau, 1861, p. 168.
[n] *Brit. & For. Med. Chir. Rev.* 1854.
[o] Barthez and Rilliet, 1853, ii. 724.
[p] *Letters to a Young Physician*, Boston, 1855, p. 326.

peritoneum is laid bare by the intestinal ulcers, the act of vomiting may induce perforation.

Laxatives. While it is the almost universal practice in this country to check the diarrhœa of enteric fever, it is not a little remarkable, that most French physicians, including Andral, Bretonneau and Louis, have recommended the frequent administration of laxatives. The treatment, which was long famous at Paris, as the method of M. De Larroque, consisted in the administration of an antimonial emetic, followed by frequent doses of calomel, castor-oil, or seidlitz-water, laxative enemata, and cataplasms to the abdomen. Diarrhœa, meteorism, and abdominal pain were not regarded as contra-indications; but, when the purging was excessive, the treatment was suspended for twenty-four hours. The practice was founded on the belief, that the typhoid symptoms of enteric fever were due to the retention of decomposing matters in the intestines.[q] Andral reported favourably on this treatment, and Louis, in the second edition of his work, gave an analysis of 38 cases, in which he had tried it, and arrived at the conclusion that it was superior to all other methods, in every form of the malady.

As already stated (page 497), it is very doubtful if the cerebral symptoms of enteric fever are due to absorption of putrid substances from the intestines; and, if they were, no amount of purging could prevent absorption taking place. Moreover, the natural history of the disease appears to me to contra-indicate laxatives. The cases recorded by Louis showed that the disease was most severe where there was the greatest purging, and in my own experience, the absence of diarrhœa has usually appeared a favourable indication (see page 480). On the other hand, I have often known a severe attack of diarrhœa, coming on spontaneously or after medicine, followed by alarming prostration. The advice given by the late Dr. Todd appears to me sound:—'Restrain 'diarrhœa and hæmorrhage in typhoid fever, and when you have 'fairly locked up the bowels, keep them so. Patients will go for 'four or six days, or even longer, without suffering inconvenience 'from this state of constipation.'[r]

Mercury has been strongly recommended in enteric fever, but I have found it both useless and injurious.

Iodine. In 1859, M. Magonty, of Paris, published a work, in which he described a new *specific* method for the treatment of enteric fever.[s] This consisted in the administration of iodine and iodide of potassium, in the form of drinks and enemata, with the object of destroying the putridity of the intestinal contents. I

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[q] Larroque, 1835. [r] Todd, 1860, p. 180. [s] Magonty, 1859.

have tried this treatment in several cases, without observing any benefit; and in one instance, it appeared to induce fatal hæmorrhage from the bowels.

Rational Method of Treatment.

We possess no specific for pythogenic fever, any more than for typhus. Baglivi's remark on 'mesenteric fever,' made two centuries ago, holds good at the present day :—'Sed quod præ cæteris 'amimadverto, in nullo morborum genere, tantâ opus est patientia, 'expectatione, cunctationeque, ad bene et feliciter medendum, 'tamquam ad bene curandum, febres mesentericas.[t] But though much mischief may be done by the *nimia diligentia medici*, by depletion on the one hand, or by over-stimulation on the other, it must not be thought that the best treatment is one of mere expectancy. Although we cannot *cure* the disease, we must *treat* it, and it is probable that many lives are saved by medical interference at the proper time, and in the proper way. In the treatment of every form of fever, and of pythogenic fever particularly, it is always important to ascertain, as nearly as possible, the duration of the disease, so that no measures be adopted, which can thwart the natural process of recovery. As in typhus, our objects in treatment must be:—1. To neutralize the poison and correct the morbid state of the blood. 2. To eliminate the poison, and the products of the destructive metamorphosis of tissue. 3. To reduce the temperature. 4. To sustain the vital powers. 5. To relieve distressing and dangerous symptoms. And 6. To avert and attack local complications.

1. Although we are ignorant of the actual nature of the pythogenic poison, it is probable that the typhoid state, which it often induces, depends on a condition of the blood similar to what is observed in typhus (see pages 265 and 497), and that it will be benefited by similar treatment. Accordingly, the mineral acids have been strongly recommended in the treatment of enteric fever by many writers,[u] and, from considerable experience in their use, I believe that there are no remedies superior to them, and that they are often of real service. Still I feel it my duty to state, that the assertion repeatedly made of late years in the medical journals, to the effect that the mineral acids constitute an almost infallible cure for typhus and enteric fever, must be founded on very limited observation. I have rarely found the acids contra-indicated by the abdominal symptoms. The hydrochloric and sulphuric acids are

[t] Baglivi, 1696, ed. 1704, p. 51.

[u] See page 265.

those which I prefer. From fifteen to thirty minims of the dilute acids may be given every three or four hours. With each dose of the acid, I am in the habit of combining about half a grain of quinine, which I believe to be of great service, especially when the disease has anything of a remittent character. Although I have never known enteric fever cut short by quinine, and have found it in large doses, as useless and injurious as in typhus, I have repeatedly observed the febrile exacerbations reduced in severity, the appetite improved, and the strength increased, under the use of the mineral acids and small doses of quinine, which may be prescribed as follows, for an adult :—

℞—Acid. Sulph. dil. . . . } ♏xv. ad xxx.
vel Acid. Hydrochlor. dil. . } ♏xv. ad xxx.
Quinæ Disulph. . . . . gr. ¼ ad gr. j.
Syrup. Aurantii . . . . ℥ss.
Aquæ Carui ad . . . . ℥j.
Fiat. haust. 3â vel 4â horâ sum.

2. Elimination is to be encouraged by maintaining the action of the kidneys and of the skin. The nitrate and bitartrate of potash, recommended in the case of typhus, are here objectionable, on account of the condition of the bowels, but nitric ether or decoction of Broom-tops may be given with impunity.

Tea and coffee, and perhaps common salt, are equally valuable remedies as in typhus, and probably act in the same way, by promoting the elimination of urea. The reader is directed to the remarks made on these remedies at page 267. Theine and caffeine, the active principles of tea and coffee, are also well worthy of trial, particularly in cases where there is much stupor.

The action of the skin is to be kept up by sponging with cool or tepid water, repeated two or three times daily.

For reasons already given, we must refrain from acting on the bowels. It may be doubted, if the diarrhœa of enteric fever is a natural process of elimination, for severe diarrhœa often co-exists with the most decided cerebral symptoms, and, in the mildest cases, diarrhœa may be absent. Moreover, the circumstance, that the quantity of urea excreted by the kidneys is not affected by the diarrhœa (page 485), shows that the bowels are not the channel for its elimination in enteric fever. The rule which I adopt, with regard to the bowels, is this. When severe diarrhœa is succeeded by constipation, I abstain from interfering for four or five days, and then I prescribe a simple enema, or one teaspoonful of castor oil. When the bowels are confined throughout, I order one or other of the same remedies at the end of every third or fourth day. Jalap, colocynth, and all the ordinary purges, are, as Baglivi long ago remarked, 'to be shunned like the plague' (see page 390).

3. Various remedies have been employed for reducing the pulse and temperature in enteric fever. Wunderlich has strongly recommended digitalis for this object; and he maintains, that it not only reduces the pulse and temperature, but that it diminishes the severity of the disease. He gives it in the form of infusion, and in doses which correspond to about five fluid ounces of the London infusion, in the twenty-four hours.[x] The veratrum viride is highly praised by American practitioners for the same object. I have had no experience of these remedies. The cold affusion is a method of acknowledged power for reducing the pulse and temperature (see pages 259 and 567), and deserves further trial. I have often observed marked relief derived from cold and tepid sponging.

4. The vital powers are to be sustained by appropriate nourishment given from the commencement of the disease, with the precautions already mentioned (p. 566). Tea and coffee ought to be prescribed several times in the day. They not only appear to promote elimination, but they act as stimulants on the nervous system, and perhaps check the destructive metamorphosis of the tissues.

When the circulation becomes feeble, we must have recourse to wine or brandy. As a rule, however, alcoholic stimulants are required much less frequently than in typhus, and not until a later period of the disease. They are rarely necessary before the tenth or twelfth day, and a large number of cases do perfectly well, if not better, without them at any stage. This remark applies especially to young children. A few years ago, I employed brandy very largely from the very commencement of the disease, and on comparing the results of this treatment, which were noted with great care, with those of my present practice, I am satisfied that brandy will not *prevent* the strength from failing, and the body from emaciating. The prostration was as early, and the emaciation as great, with the brandy, as without it (see p. 257). To *prevent* emaciation and failure of the strength, we must trust to *food*—to milk, beef-tea, and arrowroot, and reserve alcohol for the time when the circulation becomes feeble, which is usually much later than in typhus. Then it is that alcohol is invaluable. The indications for the use of stimulants are the same as in typhus (see p. 269); in both diseases, we must be mainly guided by the state of the heart and of the radial pulse.

The best alcoholic stimulants in enteric fever are sherry, old port, and brandy.[y]

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[x] Wunderlich, 1858.

[y] Brandy (1 part) and milk (2 parts), and wine-whey, which is prepared by adding one-part of sherry to two of boiling milk, and then straining, are excellent forms for giving stimulants.

In addition to wine and brandy, we may employ other stimulants, such as chloric and sulphuric ether, quinine, liquor cinchonæ, musk, and camphor, which may be combined with the remedies already recommended (see page 272). Carbonate of ammonia is still more objectionable than in typhus, as it is apt to irritate the bowels and increase the diarrhœa (page 272).

5. The chief symptom demanding interference is usually *diarrhœa*. It is always well to have recourse to astringents when there are more than two motions in the twenty-four hours, and if the patient be very prostrate, even this amount of action may be injurious.

The milder forms of diarrhœa are usually checked by administering towards evening a starch enema containing from ten to twenty drops of laudanum, and by adding two or three minims of the liquor opii sedativus to each dose of the acid medicine.

If this treatment fails, recourse may be had to the following prescription, while, at the same time, the starch and opium enemata are persisted in—

℞—Acid. Sulph. Aromat. . . . ℥xxx.
Liq. Opii. Sedativ. . . . ℥iij
Aq. Menth. pip. . . . . ℨj Misce.
Fiat. haust. 4tâ vel 6tâ q.q. horâ sum.

If the acids are not tolerated by the stomach, the acetate of lead may be tried. This has long been the favourite remedy at the London Fever Hospital. It may be given in solution, in doses of two or three grains every four or six hours, with or without an eighth of a grain of acetate of morphia. Along with the lead, the opiate enema may be given at bed-time, or a powder composed of equal parts of Dover's powder and Hydrargyrum cum creta. The acetate of lead is particularly useful in cases where opium is contra-indicated (see page 277).

In addition to these remedies, much benefit will be derived, in every case of enteric fever, from constant fomentation of the abdomen by means of linseed poultices, or, still better, by wet flannel, covered with oiled silk, or gutta percha. These applications ought to be omitted in no case where there is diarrhœa, abdominal pain, or tympanitis. Stupes, moistened with turpentine, or with the compound camphor liniment, ought also to be applied at intervals.

Other plans of treatment have been proposed, a knowledge of which may be sometimes advantageous.

Huss strongly recommends small doses of ipecacuanha, in combination with phosphoric or sulphuric acid, and fomentation of

the abdomen, and assisted, if need be, by starch and opium enemata.[z]

Some years ago, Professor Fouquier, of La Charité, spoke very highly of alum dissolved in solution of gum. He commenced with twenty-four grains in the day, and gradually increased the quantity up to a drachm[a] Alum may also be given in the form of *alum whey*, which is prepared by adding one drachm of alum to a pint of boiling milk, and then straining. Two ounces may be given after each motion of the bowels.

Trousseau commences by giving a saline laxative of sulphate of soda, or a seidlitz powder, which, in his opinion, checks the diarrhœa by altering the secretions, and is particularly useful when the diarrhœa is accompanied by much meteorism. If this does not succeed, he then orders the English mistura cretæ or equal parts (7 grains of each) of prepared chalk and of trisnitrate of bismuth, to be taken from three to eight times in the twenty-four hours. These remedies failing, he has recourse to pills containing about a tenth of a grain of nitrate of silver.[b]

Nitrate of silver has been recommended by many other practitioners, both by the mouth and also in the form of enema. In the latter form, it is difficult to understand its mode of action, seeing that the ileum is the chief seat of the disease.[c] In severe diarrhœa, after the fourteenth day, Dr. Joseph Bell has sometimes found advantage from nitrate of silver, in doses of from one to three grains made into a pill, and taken every six or eight hours.[d] Sulphate of copper is a favourite remedy with some practitioners. I have rarely employed either it or nitrate of silver during the primary fever; but I have often found these remedies most useful in the diarrhœa arising from the atonic ulcers, after the fourth week of the disease. They may be given in doses of a quarter of a grain, with a third of a grain of opium, three times a day.

The vegetable astringents, such as logwood and catechu, are sometimes useful, when opium is contra-indicated (see page 277).

Hæmorrhage from the Bowels, during the first ten days of the disease, is usually slight, and I have in most cases found it checked by the acetate of lead and morphia, and by the starch and opium enemata already recommended for diarrhœa. When the hæmorrhage is considerable, the remedies which I employ are sulphuric acid and opium, with opiate enemata. The mixture prescribed

[z] Huss, 1855. [a] *Brit. & For. Med. Rev.* 1836, i. 568.
[b] Trousseau, 1861, p. 182.
[c] Yates, 1853; also, Boudin, *Journ. des Con. Méd. Prat.* Mai, 1839.
[d] Bell, 1860, viii. 385.

above, for diarrhœa, may be given every hour, or every half-hour, and if the hæmorrhage be excessive, it may be well to add to each dose five grains of gallic acid. Tannine, rhatany, turpentine, and the perchloride of iron, are other remedies which have been found successful in checking the hæmorrhage. Turpentine was recommended by Dr. Graves, and I have tried it in several cases with advantage; but when the hæmorrhage is copious, I prefer the sulphuric acid and opium. The turpentine may be given in doses of ten to thirty minims every hour, in a mixture with mucilage and peppermint water. Along with these internal remedies, perfect rest is to be enjoined, a bladder containing broken pieces of ice is to be applied over the right side of the abdomen, and small pieces of ice may be given to suck. Stimulants are to be administered according to the state of the pulse.

For *vomiting* occurring during the first ten days of the disease, the best remedy is an emetic. If emetics fail, or if they be contra-indicated, as they always are after the tenth day (see page 568), a sinapism or a turpentine-stupe is to be applied to the epigastrium, while ice is given to suck. If the vomiting continues, which rarely happens, the acid treatment must be suspended, and lime-water, or bismuth and hydrocyanic acid are to be substituted. Equal parts of lime-water and milk I have often found an excellent remedy, in such cases. The practitioner should bear in mind, that vomiting coming on suddenly after the fourteenth day, is often the first symptom of peritonitis.

Tympanitis is sometimes so excessive, as to impede the breathing, and cause great distress to the patient. Much may be done to prevent and relieve it, by the application of turpentine stupes, and by constant fomentation of the abdomen (see page 573). If necessary, other measures must be resorted to. The gas, as we have found, is mainly contained in the colon, and accordingly enemata are the most effectual remedies. An enema of assafœtida (Tinct. Assaf. ʒij. Decoct. Hordei, Oj.) or of peppermint-water, confection of rue, or infusion of chamomile often gives great and immediate relief. If the tympanitis continues, the acetate of lead and morphia, or turpentine, as prescribed at page 286, are often useful.[e] Some practitioners recommend that the gas be drawn off by an

[e] Dr. Wood of America recommends turpentine in all cases of enteric fever, where there is tympanitis and a dry tongue. In certain cases, where the tongue, after cleaning, becomes dry, red, and, smooth (a symptom which, he thinks, indicates great danger, and, at all events, slow cicatrization of the intestinal ulcers, and a protracted convalescence), he regards turpentine almost in the light of a specific. He gives it in doses of from 5 to 20 minims every hour, or every second hour. (*Pract. of Med.* 2nd ed. 1849, i. 328).

œsophagus-tube introduced by the rectum, but I have no experience of this practice, as I have rarely found the measures already recommended fail to give relief. In the typhoid stage, tympanitis is often a sign of debility, and must be treated with stimulants.

For *abdominal pain and tenderness*, the best treatment is assiduous fomentation and poulticing of the abdomen. When this is kept up from an early stage of the disease, I believe that it often prevents the pain. When the pain is severe, opium or morphia may be given by the mouth or rectum, and the turpentine-stupe may be applied to the abdomen at intervals. When the patient is young and robust, and the fever is at an early stage, from two to six leeches applied over the right iliac region or round the anus, often give great and immediate relief (see page 567).

Epistaxis, as a rule, is slight, and requires no treatment; but when profuse, it must be checked without delay (see page 495). Gallic acid and sulphuric acid, as already prescribed for hæmorrhage from the bowels, may be given every hour, or every half-hour; a small bladder containing ice is to be applied over the forehead and nose, while a solution of alum or tannine, or an infusion of matico or rhatany, is injected into the nares. If these measures fail, the nares must be plugged.

Headache (page 273), *sleeplessness* and *delirium* (page 274), *drowsiness* and *stupor* (page 279), *convulsions* (page 284), and *hiccup* (page 286), are to be treated on the same general principles as in typhus, except that purgatives, and all remedies calculated to irritate the bowels, ought to be rigidly abstained from. In all cases where the patient is unconscious, the condition of the bladder must be attended to (see page 280).

6. Among the *complications* of pythogenic fever, the most important is *peritonitis*. As already stated, there are no means by which we can distinguish with certainty between peritonitis from perforation of the bowel, and peritonitis from other causes (see page 507); but, in the great majority of instances, perforation is the cause. The case, though desperate, is not altogether hopeless (see page 510). Opium is our only sheet-anchor in such cases; but to be of service, it must be given immediately and boldly, in full doses. To an adult, two grains of solid opium may be given at once, followed by one grain every hour, or every second or third hour, till the pain and tenderness are relieved. The precise dose must be regulated according to the age and other conditions of the patient. The amount of opium, which is tolerated under such circumstances, is sometimes truly extraordinary: as

much as sixty grains have been taken in three days, with impunity. The opium is to be given alone, and never in combination with mercury, which increases the flow of bile, and so excites the peristaltic action of the intestines. Our object is not to produce absorption of lymph, (even if mercury has such a power,) but to paralyse the bowels, so as to prevent the escape of their contents into the peritoneum, and favour the formation of adhesions. Many writers recommend the application of leeches to the abdomen on the supervention of peritonitis; but the extreme prostration of the patient, and the circumstance that the tendency is to death by syncope, have always appeared to me to contra-indicate such a practice. In most cases, relief to the pain and tension will be obtained by covering the abdomen with turpentine-stupes, alternating with warm fomentations, and thin linseed poultices. Huss recommends the application to the abdomen of a bladder of ice, a practice which appears to me well worthy of trial. At the same time, the patient is to be kept in a state of absolute immobility, and strict attention must be paid to the ingesta, which ought to be perfectly liquid, and given in such small quantities at a time, that they can be absorbed by the stomach. A table-spoonful of beef-tea, or, if necessary, of iced brandy and water, may be given every half-hour, or every quarter of an hour. The large quantities of food and stimulants, sometimes given under such circumstances, cannot fail, in my opinion, to be injurious. Dr. Joseph Bell, indeed, reports that he has treated several cases to a successful termination, with opium and absolute starvation. For the first two or three days, he allows nothing in the way of nourishment except a table-spoonful of water, or of toast-water, every quarter of an hour.[f] If the case does well, we must beware of interfering with the constipation induced by the opium; the bowels may be left to act spontaneously. Cases are recorded, where the incautious administration of a purge appeared to have broken up the adhesions and produced a fresh and fatal attack of peritonitis.

Notwithstanding the measures here recommended, peritonitis, as a rule, terminates in death; and hence it is imperative to do all in our power, *to avert its occurrence.* Bearing in mind that rupture of the denuded peritoneum is one of the ways in which perforation takes place, it is obviously of the greatest importance to prevent every movement, which would favour such a laceration. Attention to this point is particularly necessary in mild cases, in which the patient is able to get up without assistance, and in

[f] Bell, 1860, viii, 386.

which perforation is most apt to occur (see pages 508 and 520). It is a good rule never to allow the patient to get out of bed to the night-stool, after the tenth day of his illness, until convalescence is fairly established, and not even then, if there is reason to believe that the ulcers have become atonic, instead of cicatrizing. In the advanced stages of the disease, the physician ought to be very careful in the way in which he manipulates the abdomen. Purgatives are to be abjured, and the rules laid down as to diet, (page 566) are to be strictly enforced, while warm fomentations or poultices are to be applied to the abdomen, from an early stage of the disease.

Bronchitis (page 281), *hypostatic consolidation of the lungs, pneumonia* (page 283), and *pleurisy* (page 284), are to be treated in the same way as in typhus, care being taken to abstain from all remedies calculated to irritate the bowels. In cases of pneumonia, the acetate of lead is often peculiarly appropriate, as it serves at the same time to control the diarrhœa.

In cases of *laryngitis*, a small blister may be applied on either side of the neck below the angles of the jaw, while the whole neck is enveloped in a warm poultice. Gargles are of no use, but advantage will be derived from the insufflation of alum, or from the application to the orifice of the larynx of a strong solution of nitrate of silver, or of the tincture of the muriate of iron. The supervention of œdema glottidis is the danger to be apprehended; and in cases where suffocation is imminent, recourse should be had at once to tracheotomy. Several cases are recorded in which the operation has been successful.[g]

Hyperæsthesia (page 285), *partial palsy* (page 285), *mental imbecility* (page 285), *bed-sores* (page 286), *gangrene* (page 287), *erysipelas* (page 287), *phlegmasia alba* (page 288), and *inflammatory swellings* (page 289), demand the same treatment as in typhus.

Treatment during Convalescence.

Patients convalescing from pythogenic fever, require far more care and watching than those recovering from typhus. While the intestinal ulcers are undergoing cicatrization, it is obvious that much mischief may be done by purgatives and improper diet. For a month after the cessation of the primary fever, castor oil and simple enemata are the only means which should be resorted to for opening the bowels. Although it is sometimes no easy matter to refrain from yielding to the cravings of the patient's

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[g] Trousseau, 1861, p. 197.

appetite, meat ought not to be allowed for at least seven days after the commencement of convalescence, especially in cases where diarrhœa and other abdominal symptoms have been prominently developed after the fourteenth day. The diet is to be restricted to farinaceous articles, milk, eggs, custards, light puddings, beef-tea, chicken-tea, and calf's-foot-jelly. Before meat is allowed, a piece of boiled sole, smelt, or whiting, may be given for a day or two, and its effects watched. Malt liquors ought not to be given before meat, as they are apt to derange the bowels. When convalescence is slow, quinine, the mineral acids, and steel are indicated.

When diarrhœa persists during convalescence, the acetate of lead, sulphate of copper, or nitrate of silver, must be ordered according to the instructions already given, and some astringent, such as logwood, is to be given after each motion of the bowels. The patient is to be kept in bed, and the diet is to be restricted to milk, eggs, soup, and farinaceous articles.

The liability to perforation, or to a relapse during convalescence, must always be borne in mind.

Trousseau remarks, that when there is great emaciation, especially in cases which have been treated on too lowering principles, vomiting and purging during convalescence are symptoms of a purely nervous character, and are at once relieved by the exhibition of solid food.[h]

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[h] Trousseau, 1861, p. 188.

CHAPTER V.

On the Specific Distinctions of Typhus and Enteric Fever.

I PURPOSE, in this chapter, to consider the chief arguments in support of the specific distinctness of typhus and enteric fever, derived, in the first place, from their symptoms and *post-mortem* appearances, and, in the second, from their etiology.

A. *Symptoms and Post-Mortem Appearances.*

On comparing the clinical history and anatomical lesions of the two diseases, an impartial observer cannot fail to admit, that typical cases present a striking contrast. On the one hand, we have a fever of a more or less remittent type, with a definite anatomical lesion, which is characterized by lenticular rose-spots appearing in successive crops, diarrhœa, abdominal tenderness, hæmorrhages from the bowels and nose, and dilated pupils, and which lasts, on an average, between three and four weeks: on the other hand, we have a fever of a more continued character, with no definite anatomical lesion, which is characterized by a measly eruption, the spots of which often become converted into petechiæ, by a peculiar odour, and by a great tendency to stupor and contraction of the pupil, and whose average duration, when uncomplicated, rarely exceeds fourteen days. But although the clinical histories of the two diseases are in most cases widely different, there can be no doubt that certain symptoms which characterize one are sometimes absent, or are occasionally observed in the other. For example, typhus fever may be complicated with diarrhœa or hæmorrhages, or the bowels may be confined in enteric fever. Still, even in such cases, if one or other of the eruptions be present, a diagnosis as to the condition of the intestines may be made with certainty. If in the circumstances referred to, there be no eruption, the diagnosis may be difficult, but, as Louis long ago observed, the difficulty is not greater than is often experienced

in the diagnosis of other maladies, which, as a rule, are most easily distinguished.[1] Practically, I believe that the cases are rare, in which a diagnosis cannot be made; the difficulty far oftener arises in diagnosing enteric fever from other diseases, universally acknowledged to be distinct (see pages 525-8).

From my own observations, and from a careful study of the entire question, I believe that the two following propositions will be found to hold good.

1. *When lenticular rose-spots, as described at page* 468, *appear in successive crops in the course of continued fever, the abdominal lesions of enteric fever are invariably present.*

2. *When the eruption of typhus, described at pages* 117 *and* 127, *shows itself in the course of continued fever, the abdominal lesions of enteric fever are absent.*

In addition to the evidence in support of these two propositions already adduced (see pages 231-3 and 400-6, 540), it may be mentioned that during the last fourteen years, *many hundreds* of bodies have been dissected at the London Fever Hospital, without a single exception being met with.

Statements of an opposite nature have occasionally been made; but the small number of cases adduced in their support, shows at all events, that the exceptions are very rare, while, in my opinion, most, if not all of them, are liable to one or other of the following objections.

1. No definite signification has been attached to the names employed to designate the eruptions; and arguments have been founded on the *names* given to the eruption by different observers, instead of on the characters of the eruption in each case. There can be no doubt, that much confusion has arisen in discussing the question, from different observers employing the same name to designate different eruptions. Thus, one writer speaks of the characteristic rose-spots of enteric fever becoming converted into petechiæ (in a case where the intestines were healthy); a second argues as if all cases of continued fever with 'maculæ' were typhus, and as if enteric fever had no peculiar eruption; a third applies the term 'rose-spots' to the lighter eruption of typhus; a fourth uses the term '*rose-coloured petechiæ*'; while a fifth records a characteristic case of 'typhoid fever' (*sic*) with a *mulberry-rash*. This want of precision in nomenclature, accounts for some of those cases where typhus and enteric fever have been said to have a common origin. For example, a case was reported a few years since in one of the medical journals, which was thought

[1] Louis, 1841, ii. 324.

to prove to demonstration that typhus and enteric fever arose from one poison. The evidence simply amounted to this. The physician who reported the case, had under his care one member of a family, who had fever with successive crops of '*rose-spots*,' and he was *informed* by another practitioner, that a second member of the same family had fever at the same time, with a '*mulberry-rash*.' Whatever were the facts of the case, the evidence failed entirely in establishing the conclusion which was drawn from it. If the physician in question had turned to the number of the journal, immediately preceding that in which his case was reported, he would have found the account of a case of 'typhoid fever,' (*sic*) with 'marked symptoms,' one of these characteristic symptoms being '*a very distinct mulberry rash*' over the body. Again, another physician refuses to allow that there is any difference between the lenticular rose-spots of enteric fever and the eruption of typhus, and yet, in order to prove that the two fevers are the same, he records two cases in which a 'typhus eruption' was observed during life, and the anatomical lesions of 'typhoid fever' were discovered after death; but unless a definite meaning be attached to the term 'typhus eruption,' as distinct from the eruption of enteric fever, it is needless to discuss the question. As Jacquot observed concerning the only French surgeon in the Crimean army, who maintained that the lesions of dothinenteritis were found in typhus, 'Comme il confesse, qu'il ne peut distinguer 'un typhus d'une fièvre typhoïde, son assertion n'a dès lors plus 'rien d'étrange.'[k]

2. The lighter florid spots of typhus have been mistaken for the eruption of enteric fever. The spots of typhus, at their first appearance, are often slightly elevated above the surface, and disappear upon pressure, and, if they be accompanied by no mottling, as sometimes happens, it may at first be difficult to distinguish them from 'lenticular rose-spots.' But if these spots be watched for twenty-four or forty-eight hours, they become darker, and cease to disappear on pressure, and they are usually associated with mottling. These very changes characterize the eruption of typhus, and are inconsistent with that of enteric fever (see pages 129 and 471). It is not surprising, then, that the lesions of enteric fever were absent in a case reported by Dr. Kennedy, of Dublin, to the Medico-Chirurgical Society of London, where some of the spots, thought to be characteristic of enteric fever, were indistinguishable from true petechiæ, at the end of the fourth day from their first

[k] Jacquot, 1858, p. 256.

appearance.[1] It is probable that most of the cases, in which rose-coloured spots have been reported to co-exist with petechiæ, or with a mulberry rash, admit of a similar explanation. It is worth mentioning, that Huss, whose work is so often referred to, as proving the identity of typhus and enteric fever, and who speaks of the frequent coexistence of '*tâches rosées lenticulaires*' with the petechial eruption of typhus, does not allude to the florid spots which precede, and are converted into the petechiæ of typhus, and which at first may be slightly elevated and disappear on pressure. Huss's '*tâches lenticulaires*' (synonymous with his 'eruption typhoïde'), included all the spots met with in fever, which were neither petechiæ nor sudamina.[m] This fact must be borne in mind in studying his work. It sometimes happens that, in typhus, spots disappearing on pressure are found co-existing with petechiæ, but the important point is, that when any of the spots cease to disappear on pressure, or become converted into petechiæ, the eruption is not that of enteric fever, and the intestines are found to be healthy after death.

3. 'Petechiæ' have been regarded as the characteristic eruption of typhus. They are met with, however, in many diseases besides typhus; while, as before stated, the eruption of typhus may never become petechial (see page 128). Petechiæ, therefore, as strictly defined (page 129), are not the characteristic eruption of typhus; and the circumstance of their having been observed in certain cases of fever, where the intestines have presented the lesions of enteric fever after death, is no proof that typhus and enteric fever are identical.

4. Other morbid conditions of the Peyerian and solitary glands of the intestine, such as that produced by the deposit of tubercle, the shaved-beard appearance of French pathologists, or the slight enlargement which is observed in scarlet fever, small-pox, and many other diseases, and even their healthy state, have been mistaken for the specific lesions of enteric fever (see page 552). One of the most strenuous opponents of the specific distinctness of typhus and enteric fever admits, that he has sometimes found it impossible to distinguish between the lesions of the latter, and tubercular disease of the intestines, and asks if the intestinal lesions of enteric fever may not be due to the strumous diathesis? Another recent writer maintains, that the anatomical lesions of

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[1] 'At the end of the fourth day from their first appearance, some few of 'them I could not distinguish from genuine petechiæ. These were of a much 'larger size, darker colour, and more irregular outline than the rest.' *Med. Times & Gaz.* March 17th, 1860; and *Lancet*, March 24th, 1860.

[m] Huss, 1855, p. 86.

typhus and enteric fever are identical, and that, in fact, there is no such thing as typhus without the intestinal lesions of enteric fever. The contrary statements of many distinguished observers, are met by the assertion, that the lesions are sometimes so insignificant as to require a lens for their discovery. The lesions of enteric fever, however, require no lens for their discovery at the earliest date at which death ever occurs.[n]

Tried by the above four tests, I believe it will be found, that the cases are extremely rare which, in any way, impugn the correctness of the propositions laid down at the commencement of this chapter.

At the same time, I am prepared to admit, that, in certain very rare cases, the eruptions of typhus and enteric fever may co-exist, and that if death takes place under such circumstances, the lesions of enteric fever will be found in the bowel. But if such rare cases are employed to prove the identity of typhus and enteric fevers, the same line of argument would necessitate the conclusion that all the acute specific diseases spring from one poison,—that, in fact, small-pox, scarlet fever, and enteric fever, are the same disease. It has already been shown in this work, that enteric fever and scarlatina, and also variola and typhus, occasionally co-exist in the human system. The facts recorded by Marson,[o] and many other observers, prove beyond doubt that persons may be attacked at the same time by variola and scarlatina, and I have elsewhere collected numerous instances, demonstrating the contemporaneous existence in the system of almost any two of the diseases, which are believed to spring from different specific poisons.[p] If one poison can generate a certain group of symptoms, and a second poison generate another group, a combination of the two poisons may give rise to a third group of symptoms, partaking of the characters of the two first, without necessitating the conclusion, that the first two groups of symptoms are merely different manifestations of the same poison. Some years ago, I published the reports of three cases, in which the patients appeared to suffer simultaneously from both typhus and enteric fever, in consequence of exposure to the poisons of both diseases.[q] In the two following instances, patients contracted typhus in the London Fever Hospital, while still suffering from enteric fever, for which they had been admitted :—

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[n] I have employed a lens in examining the intestines of a large number of cases of typhus fatal after the tenth day, but I have never discovered anything in the least resembling the lesions of enteric fever, such as I have seen as early as the second or sixth day (see page 541). Barrallier also employed a lens, and came to the same conclusion. (Vide *antea*, p. 233).

[o] *Med. Chir. Trans.* vol. xxx. [p] Murchison, 1859 (4). [q] Ibid. p. 197.

Case XXXIX.

Co-existence of Typhus and Enteric Fever.

George B—, aged 14, was admitted into the Fever Hospital, under the care of Dr. Buchanan, on June 3rd, 1862. He had been ill for ten days; and on admission, he presented all the ordinary symptoms of enteric fever. Pulse 108. Tongue moist and fissured; belly blown and tender over cæcum; 2 watery stools. Several well-marked lenticular rose-spots. Two of this boy's sisters had been admitted about a fortnight before, with the same fever, which ran the usual course. Both had lenticular rose-spots, and one died of intestinal hæmorrhage. The boy was placed in a ward, in which were many patients suffering from typhus.

On June 12th, fresh lenticular spots were noted as appearing; skin warm and dry. Tongue moist and fissured; belly tender; bowels still relaxed. Pulse 80. Intelligence clear. Slight headache.

June 17th, (about 22nd day). Tongue red and dry in the centre; belly tender and tympanitic; one stool. Lenticular spots still distinct, and trunk covered with a dusky mottling like that of typhus. Pulse 90. Headache much increased.

June 18th. The mottling had developed itself into an unmistakeable typhus rash, in the midst of which could be singled out a few pink, circular, elevated, lenticular rose-spots of enteric fever, which had been encircled with ink.

June 19th. Pulse 90. No motion for two days. Expression stupid, and is a little confused when spoken to.

June 20th. The mottling has faded a little, but is still distinct. Several fresh lenticular spots. To day the patient was visited by Dr. A. P. Stewart, who was satisfied with the existence of both the eruptions. Bowels still confined.

June 21st. One fresh lenticular spot; mottling remains. No fresh lenticular spots appeared after this date; but the mottling was still visible on June 26th. The patient was discharged well, on July 8th.

Case XL.

Co-existence of Typhus and Enteric Fever.

Henry W——, aged 25, was admitted into the London Fever Hospital on January 10th, 1853, having been taken ill on the 6th. He was brought from Croydon, ten miles distant from London, where enteric fever (but not typhus) had been very prevalent for some months (see page 441). Another patient suffering from enteric fever was admitted at the same time, from the same house. There were several cases of typhus in the wards, in which these patients lay. The chief symptoms in Henry W——, were giddiness, headache, vomiting, flushing of the face, disturbed sleep, loss of appetite, thirst, a variable pulse, and lenticular rose-spots. Several of these spots were noted as late as February 2nd (twenty-eighth day). On January 30th (twenty-fifth day), the patient complained of

irregular chills, alternating with flushing. The headache returned; and the tongue, which a few days before had been clean and moist, became coated. The pulse which for several days had never exceeded 72, rose to 86. For the next few days, the patient complained greatly of headache, pains in the limbs, thirst, and diarrhœa. On Feb. 4th, the pulse was 120, and the body was covered with a well-marked typhus-eruption consisting of spots and mottling. The bowels were moved ten times. The rash continued very copious for ten days, and all this time the bowels were much relaxed. On February 5th, there was copious epistaxis. There was occasional delirium, and for five days the pulse remained at 132, without any variation. Between the fortieth and forty-first days, or about the fourteenth day of the attack of typhus, the pulse fell from 120 to 72, and from this date the patient convalesced rapidly.

In the following case, the patient appeared to be exposed to the poisons of both typhus and enteric fever, before admission into the hospital.

Case XLI.

Supposed Co-existence of Typhus and Enteric Fever.

Norah H——, aged 16, was admitted into the London Fever Hospital, under my care, in December 1857, on the eighth day of an attack of fever. Her body was covered with a well-marked typhus-eruption, composed of spots and mottling, and she presented all the ordinary symptoms of typhus, viz., a dry, brown tongue ; confined bowels ; heavy confused expression ; small pupils and low wandering delirium. On the eleventh day, the typhus eruption faded, and was succeeded by lenticular rose-spots, which came out in successive crops for more than a week, and were accompanied by diarrhœa, abdominal tenderness, red tongue, and dilated pupils. A fortnight before this girl's admission, she had slept away from home, in the same bed with another girl who had 'fever.' The father and brother of this second girl were admitted into the Fever Hospital with well-marked typhus. On the other hand, it was ascertained that in the house where Norah H—— lived, the drainage was very bad, and that the water-closet had been greatly neglected, so as to become most offensive.

From these observations, I think it is evident, that the occasional co-existence of the eruptions of typhus and enteric fever, or even the discovery in rare cases of the lesions of enteric fever, in a case where a typhus-eruption has been observed during life, is no proof that the two fevers in question spring from one poison. These occurrences are very rare exceptions to a general rule, and are, in fact, not more common than the co-existence of enteric fever and scarlatina, or of scarlatina and variola.

The observations made by Landouzy, on the epidemic in the jail of Rheims, in 1839-40, which have been so often referred to as

proving the identity of typhus and enteric fever, must be viewed in connection with the cases now narrated. The fever attacked 138 persons, of whom 17 died. It resembled typhus in being very contagious, in its short duration, and in being characterized, in most cases, by constipation, congestion of the conjunctivæ, great stupor, the early occurrence of delirium, a mousy odour, and the presence of the typhus-eruption. It also attacked those persons who had previously had enteric fever, but spared those who had suffered from typhus twenty-five years before. On the other hand, it is stated, that in some of the cases lenticular rose-spots were mixed up with the eruption of typhus. This statement is fairly open to the objection, that Landouzy did not admit that the spots of typhus ever disappeared on pressure, and that he seemed to doubt if the spots of this nature, described by Stewart as existing in typhus, were not lenticular rose-spots.[q] In 6 of the 17 fatal cases, however, the intestines were examined, and were said to present the lesions of enteric fever. Two only of the 6 cases are recorded, with regard to which it has been justly observed, that the lesions were not so characteristic of enteric fever as Landouzy stated.[r] In one of the two cases, they were so slight, that Landouzy's three colleagues, and the pupils, after opening the intestine, and examining it most carefully, exclaimed, at first, that to their great surprise, they could find no disease.[s] But even admitting the facts, as stated by Landouzy, they do not prove the identity of typhus and enteric fever. Bartlett, in the first edition of his work, suggested that the causes of the two fevers were commingled at Rheims, and that a hybrid progeny was the result.[t] It is generally admitted, that the causes of enteric fever are in constant operation throughout France, while it has been shown that the end of autumn (when the epidemic at Rheims commenced) is the season most favourable to these causes being called into action. On the other hand, the prisoners were exposed to the recognized cause of typhus. The origin of the epidemic, in fact, was attributed to over-crowding (*vide antea*, page 105). It is remarkable, that Landouzy himself arrived at the conclusion that

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[q] Landouzy, 1842, p. 325.

[r] 'We doubt whether M. Landouzy has made out his case; the alterations he 'signalizes are not sufficiently defined, to bring them within Louis' definition 'of typhoid fever,' (*Brit. & For. Med. Chir. Rev.* July, 1851). 'The appearances 'Landouzy describes are perfectly well-known to the German observers, as 'dependent on a catarrhal condition of the mucous membrane, which may 'occur in typhus, as in pneumonia, or any acute disease. No accurate observer 'could confound this with typhoid deposit in and under Peyer's patches' (Flint, 1852, p. 243).

[s] Landouzy, 1842, p. 316. [t] Bartlett, 1842.

the fever at Rheims was *not identical* with the ordinary typhoid fever of France, although his observations have been so often quoted in corroboration of an opposite opinion.

B. *Etiology.*

Still more conclusive arguments, in favour of the specific distinctness of typhus and enteric fever, are derived from a study of their etiology (see *Introduction*, page 5).

1. *The two fevers have no community of origin.* One fever does not give rise to the other. If typhus and enteric fever sprang from the same poison, it ought to be a matter of daily observation to see one fever propagating the other, and the two fevers prevailing together in the same family or house. But experience is opposed to anything of the sort.

In 1849, Dr. Jenner showed, as the result of an investigation of the cases of fever admitted into the London Fever Hospital during three years, that, when continued fever broke out in a family or house, all the cases were typhus, or all were enteric fever, the two fevers never prevailing together; and hence he argued, that their specific causes must necessarily be absolutely different.[u]

My own experience is in accordance with Dr. Jenner's. I have repeatedly known from ten to six members of one family, whose ages varied from two to upwards of sixty, attacked with typhus, every one having the characteristic rash. And so with enteric fever; if one case in a family has been enteric fever, all have been enteric. Neither age, sex, temperament, nor any individual peculiarity, has in the least affected the form of fever. I have ascertained, from the Register and Case Books, that 3,506 cases of typhus, and 1,820 cases of enteric fever, were admitted into the London Fever Hospital during ten years. Very frequently, many members of one family, or residents in one house, were admitted at the same time, but cases of typhus and enteric fever were never brought from the same house, except after the lapse of many months, or even years. There were two exceptions which deserve particular notice, as indicating the caution necessary in investigating such cases. One of them was mentioned by Dr. Jenner: A boy, aged 16, was admitted on September 19th, 1848, with enteric fever, and on October 10th, his father was admitted with typhus. But the mother of the boy had visited him in the Fever Hospital, whence she might have carried the poison of typhus to her husband. The father, moreover, had been little exposed to

[u] Jenner, 1849 (1).

the contagion emanating from the son, who, being a vagabond at variance with his father, *was from home when he was taken sick.* The circumstances of the other exceptional case were as follows:— In November and December, 1851, four servants were admitted from a hotel in the Haymarket, all with enteric fever, and in the following January, a servant was admitted from the same house with typhus. This typhus patient, however, was one of the same four who had been admitted in the previous year with enteric fever. She had only left the Fever Hospital about ten days previous to her re-admission; and she had no doubt contracted typhus there, during her convalescence.

Dr. Peacock and Dr. Wilks have invariably found the same rule to hold good at St. Thomas's, the Royal Free, and Guy's Hospitals.[x] Dr. A. P. Stewart assures me, that during nine years he has never met with a single exception at the Middlesex Hospital.

These observations are not confined to London. In 1842, Dr. John Reid pointed out that all the cases of enteric fever, which had been dissected in the Edinburgh Infirmary during three and a half years, had been brought from Linlithgow, Anstruther, and other places in Fifeshire, and not one from Edinburgh itself, where typhus had been very prevalent; while Professor Goodsir showed, as the result of numerous *post-mortem* examinations, that all the cases of fever which he had observed at Anstruther during five years, had been enteric.[y] In 1846-7, enteric fever was unusually prevalent at Edinburgh, but it was shown by Drs. Waters[z] and W. Robertson,[a] that the cases occurred in the neighbourhood of Edinburgh, or in the better parts of the New Town, where typhus was scarcely ever observed. Dr. W. T. Gairdner's experience, during ten years, has been the same. In 1860, he wrote thus:— 'In Edinburgh, Dr. Begbie and myself probably have seen, or 'have had the means of knowing about, very nearly all the fever 'cases; and, therefore, when I declare to you, that, within my ex- 'perience for ten years past, no instance has occurred of a decided 'origin of enteric fever in a group of typhus cases, or of typhus 'fever in a group of enteric cases, I am entitled to say, that I 'have obtained very strong evidence in corroboration of the views 'that these two diseases are, in reality, different diseases, and not 'mere varieties of the same disease. Last summer, I made a very 'careful survey of the whole fever-field of Edinburgh (if I may 'call it so), for several months together. It was not an epidemic

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[x] Peacock, 1856 (1); Wilks, 1855.
[y] Reid, 1840 and 1842. [z] Waters, 1847. [a] Robertson, 1848.

'season; but I gathered about thirty cases of typhus, and twelve 'of enteric fever, and into the whole details of these I inquired 'with the greatest possible minuteness, visiting every one of the 'fever localities, except one or two, in which I was quite sure the 'cases were isolated. The result was, that in no case could I 'light upon a suspicion that typhus had given rise to anything 'but typhus; or enteric fever, to anything but enteric fever.'[b]

Dr. A. P. Stewart's experience in Glasgow during 1836-37-38, was of a similar nature. He tells me that he saw upwards of 3,000 cases of fever during that period, but that he had never known one instance of typhus and enteric fever occurring together in the same family or house.

Such is the nature of the evidence, from which it is inferred, that typhus and enteric differ in their mode of origin. A few observations of an opposite nature have been recorded; but in all the instances with which I am acquainted, the observers of the facts refuse to acknowledge that typhus and enteric fever can be distinguished during life, while one physician, who says that he has met with numerous instances of 'typhus' and 'typhoid fever' occurring together in the same family, maintains not only that the eruption of the one fever is convertible into that of the other, but that the anatomical lesions of the two fevers are identical, and that, in fact, there is no such thing as typhus fever, without the intestinal lesions of enteric fever. The argument against the specific distinctness of the two fevers, that they cannot be distinguished by their symptoms during life, is perfectly intelligible; but it is difficult to understand, what writers, holding this view (or, still more, holding the view that the anatomical lesions of the two fevers are identical), imply by 'typhoid fever' as distinct from 'typhus,' when they say that they have often met with examples of these two fevers at one time in the same family. It appears to me, that this is attempting to prove too much.

With one exception, recorded by Huss, I know of no instance of fever breaking out in a family, *in which the intestines have been found healthy in one member, and presenting the anatomical lesions of enteric fever in another.* Such occurrences ought not to be rare, if the two fevers so often occur together, as has been stated. Moreover, exceptional cases of the nature alluded to do not prove that typhus and enteric fever spring from the same poison, unless it can be shown that they occur more frequently than examples of the simultaneous occurrence, in the same family, of other specific

[b] GAIRDNER, 1859 and 1860.

diseases, which are universally acknowledged to be distinct. I have, on several occasions, known typhus and scarlatina, typhus and small-pox, or enteric fever and scarlatina, occur at the same time, in the same house. Dr. Jenner also mentions instances where typhus and measles, or typhus and scarlatina, or enteric fever and scarlatina, prevailed together in the same house.[c] Little weight would be attached to an opinion founded on such cases, to the effect that typhus, scarlatina, measles, and variola are all one disease.

There are several ways, in which the contemporaneous prevalence of typhus and enteric fever in the same house can be accounted for. For example, patients with enteric fever, admitted into a hospital where there is typhus, may carry the poison of the latter to their own homes, and thus become foci for the propagation of typhus, at places where there had only been enteric fever previously. In the case of large towns, where the two fevers so often prevail at the same time, it is surprising (even on the supposition that the poisons are distinct), that they occur together in the same house, so rarely as is found to be the case.

2. *An attack of one fever confers an immunity from a subsequent attack of itself, but not of the other.* While it is generally admitted, that it is extremely rare for the same individual to have two attacks of either typhus or enteric fever (see pages 92 and 436), there are numerous examples of persons contracting both of these fevers, under favourable circumstances. It has repeatedly happened at the London Fever Hospital, that patients, after having convalesced from an attack of enteric fever, have contracted typhus while still in hospital, or have been re-admitted with it, within a week or ten days of their discharge; while, in rarer cases, an attack of typhus has been succeeded by enteric fever. In fatal cases of enteric fever, following typhus, the recent abdominal lesions of enteric fever are found in the dead body; but when the sequence of the fevers is reversed, we find only the cicatrices of the old intestinal ulcers.

Similar observations have often been made elsewhere. During the summer of 1859, 12 cases of enteric fever were admitted into the Edinburgh Royal Infirmary under the care of Dr. W. T. Gairdner. There were cases of typhus in the same ward. After a distinct convalescence, 4 of the 12 cases were attacked with typhus, and 1 died of it: the eruptions in the two fevers were most characteristic.[d] Dr. H. Weber and Dr. Gueneau de Mussy have each communicated to me a case, in which an attack of enteric

[c] JENNER, 1849 (1). [d] GAIRDNER, 1860.

fever was followed by typhus: one of these cases occurred at London, the other at Dublin. Two similar cases have been observed by Gresinger at Zurich;[e] one by Godélier, at Paris;[f] one by Anderson, at Glasgow;[h] and four are mentioned by Flint[i] and Bartlett,[k] as having been observed in America. Many similar observations were made by Barrallier, at Toulon,[l] and by the French officers in the Crimean army.[m] M. Baudens communicated to the French Academy the cases of two French surgeons, who died of typhus in the Crimea, in whose intestines were found the cicatrices resulting from an attack of enteric fever, from which they had suffered four or five years before.[n] On the other hand, Corrigan records the case of a patient recovering from typhus at Dublin, who was seized with enteric fever and died;[o] a similar case is mentioned by Bartlett;[p] and Jacquot alludes to several instances, in which soldiers, who had passed through typhus in the Crimea, were shortly after attacked by enteric fever in France.[q]

The following cases illustrate the sequence of the two fevers alluded to:—

Case XLII.

Enteric Fever followed, nearly three months after the commencement of Convalescence, by a fatal attack of Typhus.

Francis B——, aged 34, was admitted into the London Fever Hospital, October 14th, 1854. This man had all the symptoms of a very severe attack of enteric fever. The lenticular rose-spots were numerous, most characteristic, and came out in successive crops. The pulse varied between 72 and 108. There was a circumscribed pink flush on both cheeks. The tongue at first red and glazed, ultimately became dry and brown; the abdomen was greatly swollen and tender; there was diarrhœa and hæmorrhage from the bowels, acute delirium, and much tremor. After the attack had lasted fully a month, convalescence commenced about the 5th of November, but was retarded by extensive bed-sores. On November 17th, he was put on full diet, with a chop and a pint of porter.

The patient was kept in hospital on account of the bed-sores, but was making a good recovery, when about the 29th of January, 1855, he lost his appetite, and felt generally unwell. On February 3rd, a faint, red, mottled typhus-eruption made its appearance on the chest. On the 4th, this eruption extended over the trunk and arms, and became much deeper in colour, and it did not disappear on pressure. There was great somnolence, but no abdominal symptoms. On February 8th, the pulse was above 150, feeble, and irregular; respirations 36, with much dyspnœa

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[e] *Med. Times & Gaz.* Dec. 21, 1861.
[f] Godélier, 1856, p. 887.
[h] Anderson, 1861, p. 116.
[i] Flint, 1852, pp. 314, 342.
[k] Bartlett, 1856, p. 293.
[l] Barrallier, 1861, p. 105.
[m] Jacquot, 1858, p. 225.
[n] Barrallier, 1861, p. 105.
[o] Corrigan, 1853, p. 91.
[p] Bartlett, 1856, p. 294.
[q] Jacquot, 1858, p. 225.

At 10 p.m. of the 11th, or about the 14th day of the attack of typhus, the patient died.

Post-Mortem Examination.

Extreme emaciation. Extensive bed-sores over sacrum and hips, and on outer aspect of both ankles. Livid patches on knees, simulating commencing gangrene.

A considerable amount of sub-arachnoid serosity. There were six drachms of serum at the base of cranium, and 1½ drachm in the lateral ventricles. Over the whole of both hemispheres was a delicate layer of coagulated blood, of a bright red colour, within the cavity of the arachnoid.

In the chest, there was hypostatic consolidation, with well-marked lobular pneumonia, of both lungs, and recent pleurisy on the left side.

In the lower 22 inches of the ileum, there were distinct traces of the old ulcers in Peyer's patches, but not the slightest evidence of recent disease. There was no deposit in the solitary glands, and the patches were not at all elevated or thickened, but most of them contained two or more smooth depressed spaces, about a quarter, or a sixth, of an inch in diameter. Their edges were nowhere thickened, but were gradually bevelled off, and many of them were covered with mucous membrane, which moved freely upon the subjacent coats. Some of the depressions had more defined edges, and the membrane covering them was adherent. There was no puckering round any of them. In the large intestine were a few isolated depressions of a similar nature. None of the mesenteric glands were at all enlarged.[r]

CASE XLIII.

Enteric Fever, followed, five weeks from the commencement of Convalescence, by an attack of Typhus. Recovery.

Jane R——, was taken ill on September 27th, 1856, with head-ache, giddiness, and cold shivers; and on October 6th, she was admitted into the London Fever Hospital. The symptoms after admission were as follows: lenticular rose-spots appearing in successive crops; pulse varying from 108 to 120; diarrhœa; occasional vomiting; much pain and tenderness of abdomen, and tympanitis. There was no delirium, and the mind was clear throughout the attack. The above symptoms lasted till about October 20th (upwards of three weeks), about which date, the patient became convalescent. At the end of the month, she was able to get up; and for some weeks she assisted the nurse in the duties of the ward, in which there was a large number of patients suffering from typhus.

On November 24th, she was taken ill with pains in the head and limbs, and loss of appetite. On November 29th, her symptoms were: pulse 132; great prostration; headache; little sleep; chest and abdomen covered with a distinct, mottled, florid typhus-eruption; tongue thickly

[r] I have notes of another case, in which the history and *post-mortem* appearances resembled those of Case XL., in all the more important particulars.

coated; bowels confined. On December 1st, pulse 130; much moaning in sleep; eruption darker. On December 3rd, pulse 126; some of the spots distinctly petechial; countenance heavy and confused; conjunctivæ injected; occasional low delirium. The patient continued much in the same state until December 8th (or the 15th day), when the pulse fell to 86, the tongue was clean, and there was copious perspiration. From this date, she rapidly improved, and on January 1st, 1857, she was discharged well.[8]

CASE XLIV.

Typhus. Convalescence on fourteenth day. After an interval of three weeks, Enteric Fever lasting about nineteen days.

Phœbe D——, aged 21, was admitted into the London Fever Hospital, on July 3rd, 1857. Six other members of the same family, the youngest aged 8, and the oldest 50, were admitted on the same day with well-marked typhus; all had the rash well-developed.

Phœbe D—— was taken ill five or six days before admission, with headache, pains in the limbs, loss of appetite, and chilliness. On July 4th, her pulse was 108; she had been very noisy and delirious during the night; expression stupid; face dusky; conjunctivæ injected; copious measly typhus-eruption, not disappearing on pressure; tongue thickly furred, but moist; bowels opened by medicine.

July 7th, pulse 108; tongue dry and brown; bowels very confined; rash much darker, and some of the spots are converted into petechiæ; still very delirious.

July 11th. Pulse 96. Patient feels and looks much better since yesterday; tongue moist; rash scarcely visible.

On July 15th, she was ordered full diet with meat; on the 20th, she was able to get up, and by the end of the month, she had almost regained her strength.

On August 3rd, the patient lost her appetite, and suffered from headache; and, after two or three days, diarrhœa came on, the motions being watery and pale-yellow.

August 8th. Pulse 100; slight headache; slept badly; tongue moist, very red at the tip and edges, with white fur along centre; three watery motions.

On August 13th, pulse was 120; and several lenticular rose-spots were observed on the chest and abdomen; great tympanitis and tenderness of abdomen; four watery stools; no delirium, and intelligence clear. Fresh rose-spots continued to appear until the 19th. On the 21st, the pulse was 96; the tongue was cleaner; the appetite was beginning to return; and the diarrhœa had ceased. From this date, convalescence advanced slowly but steadily.

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[8] I have notes of five other cases, in which the history was similar to that of Case XLI.

3. *The two diseases differ in their contagious properties, and in their mode of propagation* (see pages 80 and 430, and pages 84 and 433).

4. *The two diseases differ in their mode of prevalence.* Typhus prevails in the epidemic form; enteric fever is an endemic disease, or its epidemics are circumscribed (see pages 54 and 412, and Diagram VII.)

It has been argued that the two fevers are identical, because examples of enteric fever are occasionally met with during epidemics of typhus; but, as a rule, enteric fever is equally prevalent, when there is no typhus (see page 413). Even an increased prevalence of enteric fever during an epidemic of typhus, may be accounted for by a co-existence of the causes of the two diseases, in an unusual degree (see page 419). In the autumn of 1858, enteric fever and scarlatina were both epidemic at Windsor;[t] but no one would conclude from that circumstance, that these two diseases are identical.

Moreover, it is found that epidemics of typhus always commence among the poorest and most crowded of the population, and do not spread among the rich, whereas enteric fever attacks rich and poor alike, and often commences in the best and most ventilated localities.

5. *The increased prevalence of enteric fever, after a long continuance of hot weather, does not hold good with regard to typhus* (see pages 64 and 416–19).

6. *While the poison of typhus appears to be generated by over-crowding of human beings with deficient ventilation, that of enteric fever is contained in the emanations from certain forms of putrefying organic matter* (see pages 95 and 438).

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[t] Murchison, 1859 (3).

CHAPTER VI.

Simple Continued Fever or Febricula.

Section I.—Definition.

A SPORADIC, non-contagious disease, arising from exposure to the sun, fatigue, surfeit, inebriety, etc. Its symptoms are: frequent, full, and often firm pulse; white tongue; thirst; constipation; high-coloured urine; hot and dry skin; no eruption; severe headache, and sometimes acute delirium; the fever subsiding in from one to ten days, with copious perspirations, herpetic eruptions, etc.; rarely fatal in Britain, except from complications; but, when death occurs, no specific lesion.

Section II.—Nomenclature.

1.—*Names derived from its continued character.*

Σύνοχος? (*Greeks*); Synocha vel Synochus Simplex (*Riverius*, 1623; *Hoffmann*, 1700; *Juncker*, 1736; *Burserius*, 1785); Synocha (*Sauvages*, 1759; *Linnæus*, 1763; *Cullen*, 1769); La Fièvre Synoque (*Davasse*, 1847); Synoshische (*Germ.*); Febris continua simplex (*Lieutaud*, 1776); Simple Continued Fever (*Modern Writers*).

2. *From its supposed Inflammatory or Ardent Character.*

Συνεχὴς φλεγματώδες? (*Greeks*); Febris Sanguinea? (*Avicenna*); Synocha sanguinea? (*Sennertus*, 1641); Feb. acuta sanguinea (*Hoffmann*, 1700); Acute Continual Fever (*Langrish*, 1735); Simple Inflammatory Fever (*Huxham*, 1739; *Fordyce*, 1791); Febris acuta simplex (*Storck*, 1741); Synocha plethorica and Ephemera plethorica (*Sauvages*, 1763); Febris continens inflammatoria simplex (*Selle* 1770); Febris acuta (*Ploucquet*, 1791); Entzündungsfieber and Entzündliche Fieber (*Reil*, 1794, etc.); La Fièvre angioténique (*Pinel*, 1798); La Fièvre angioténique pure et simple (*Bouillaud*, 1826); Fièvre inflammatoire (*French*); Febbre inflammatoria (*Ital.*).

Καῦσος (*Hippoc.*); Causus sive Febris ardens (*Galen; Willis*, 1659; *Boerhaave*, 1738); Synochus causonides (*Forestus*, 1591; *Mangetus*, 1695); La Calentura? (*Piquer*, 1751); Causos (*Vogel*, 1764); Enecia Cauma (*Mason Good*, 1817); Ardent Fever (*Burnett*, 1812; *Ranald Martin*, 1841; *Copland*, 1844); Ardent continued Fever (*Morehead*, 1856).

3.—*From the absence of Putrid or Typhoid Symptoms.*

Synochus imputris? (*Galen*); Febris continua non putrida (*Lemmius*, 1563; *Boerhaave*, 1738; *Quarin*, 1781); Synocha sine putredine (*Sennertus*, 1641); Synocha non putris (*Bellini*, 1732).

4.—*From its Duration.*

Febris septimanaria (*Platerius*, 1656; *Sprengel*, 1814); Ephemera plurium dierum (*Sennertus*, 1641; *Juncker*, 1736); Synocha septimo die soluta (*Hoffmann*, 1700).

Febris ephemera (*Riverius*, 1623; *Sennertus*, 1641); *Sauvages*, 1759); Diary Fever (*Strother*, 1728); Ephemera simplex (*Boerhaave*, 1738); Febris diaria, (*Juncker*, 1736; *Linnæus*, 1763); Fièvre éphémère (*Davasse*, 1847); Febricula (*var.*, and *Jenner*, 1849, not the Febricula of *Manningham*, see page 391); Das entägige Fieber (*Germ.*); Effimero (*Ital.*); Efemera (*Span.*).

5.—*From its Causes.*

Ephemera a frigore and E. a calore (*Sauvages*, 1759); Sun Fever (*Scriven*, 1857).

Section III.—History and Etiology.

Simple Continued Fever has been referred to by many authors from the time of Hippocrates to the present day. For example, Riverius described several varieties of a '*febris simplex*,' arising from non-specific causes. Concerning one of them, '*ephemera*,' he observed: 'Ephemera plerumque generatur a causis externis; 'intra viginti quatuor horas, plerumque solet terminari.' From this, he distinguished '*synochus simplex*,' arising from the same causes, and presenting the same symptoms, but lasting from four to seven days.[u] Sennertus also distinguished between '*ephemera*' and '*ephemera plurium dierum*.'[x] Strother, in our own country, described a '*diary fever*,' as distinct, on the one hand, from '*spotted fever*' (typhus), and, on the other, from '*slow fever*' (enteric). The diary fever, he said, resulted 'from hard drinking, or two 'great heat of the sun, or from a little cold'; it needed 'little 'help from physic'; and it did 'not last above three or four days.'[y]

Unlike the fevers already considered, Simple Continued Fever is independent of any specific poison, and is, therefore, not contagious. Its ordinary causes are, exposure to great heat or cold, surfeit or inebriety, gastric derangement, imperfect excretion, and mental or bodily fatigue. Many cases are designated Simple Fever or Febricula, which are in reality mild cases of typhus or pythogenic

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[u] Riverius, 1648, ed. 1690, p. 421. [x] Sennertus, 1619.
[y] Strother, 1729, p. 159.

fever, or relapsing fever without the relapse, or catarrh, with an unusual amount of febrile disturbance. The typhus and pythogenic poisons occasionally give rise to symptoms so mild and indefinite in their nature, and of so short duration, that an accurate diagnosis is impossible, unless well-marked cases of either fever occur in the same house at the same time. Accordingly, the term Simple Fever becomes a refuge for many cases of an uncertain character. This circumstance, coupled with the fact that Simple Fever is characterized by no peculiar eruption or anatomical lesion, and that it rarely proves fatal in this country, except when some complication supervenes, to which the febrile symptoms are naturally referred, has induced some observers to doubt the existence of Simple Fever, as distinct from the fevers already described. Dr. Tweedie, for example, in his Lumleian Lectures, expressed the opinion, that all cases of so-called 'Febricula' were mild cases of typhus or relapsing fever, and did not think 'that a new nosological term should 'be introduced merely to accommodate such cases.'[z] From what has been stated, it is obvious that the recognition of Simple Fever is not a modern innovation. I am satisfied, also, that cases of short fever, independent of any specific poison, are occasionally met with in this country; while in certain parts of the world, where typhus and relapsing fever are unknown, Simple Continued Fever is a very common disease. From my own observations in India and Burmah, I feel convinced that the Common Continued Fever, the Ardent Fever, and the Sun Fever of the tropics, are nothing more than severe forms of the Simple Fever or Febricula of Britain. For additional evidence as to the existence of such a disease as Simple Continued Fever, reference may be made to the excellent monograph of Davasse,[a] who, like Riverius and Sennertus, distinguishes between a *Fièvre Éphémère* and a *Fièvre Synoque*, according to the duration of the malady; to the published lectures of Dr. W. Jenner[b] and Dr. Lyons,[c] and to various works on tropical diseases, particularly those of Morehead[d] and Sir Ranald Martin.[e]

Simple Continued Fever is a sporadic disease, and does not prevail as an epidemic in temperate climates. Philippe Ingrassia, of Palermo, however, described an epidemic of Simple Fever, which prevailed in Sicily, in 1557. It commenced with rigors, which were followed by burning heat, violent headache, flushing of the

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[z] TWEEDIE, 1860, p. 415.
[a] DAVASSE, 1847.
[b] JENNER, 1850, xxiii. 312; and 1853, p. 417.
[c] LYONS, 1861, p. 53.
[d] *Clin. Res. on Dis. in India*, 2nd ed. 1860, p. 162.
[e] *Influence of Tropical Climates*, 1856, p. 204.

face, vertigo, quick, full, and firm pulse; the symptoms subsided after four days, and the disease, although alarming, was far from being fatal: bleeding was the sole remedy. A similar epidemic, but of a more severe character, was observed by Hoyer, at Mulhausen, towards the end of the summer of 1700.[f] These epidemics closely resembled the Ardent Continued Fever of the tropics, and may possibly have been due to similar causes. In India, the Ardent Fever often assumes an epidemic form during the hot dry season.

The number of cases of Simple Continued Fever admitted into the London Fever Hospital, and the Glasgow Royal Infirmary, since 1847, is given in the following table:—

TABLE XLIV.

Years.	Lond. Fever Hospital.	Glasgow Roy. Infirmary.	Years.	Lond. Fever Hospital.	Glasgow Roy. Infirmary.
1848	91	15	Brot. forward.	775	926
1849	79	60	1856	89	43
1850	62	41	1857	72	72
1851	56	167	1858	44	78
1852	129	102	1859	34	49
1853	152	270	1860	32	37
1854	144	167	1861	49	54
1855	62	104	1862 To June 30	58	—
Total.	775	926	Total.	1153	1259

Of 1,095 cases of Simple Continued Fever admitted into the London Fever Hospital, during fourteen years, 562 were males, and 533 females. (See page 61).

Young persons and adults appear to be more liable to Simple Continued Fever, than persons advanced in life. The mean age of 845 cases, admitted into the London Fever Hospital during ten years (1848-57), I ascertained to be only 22·82, which is about three years less than the mean age of the entire population (see page 62). Of the total number, 789, or upwards of 93 per cent., were under forty-five. In the tropics, it is the young and robust, and persons newly arrived from temperate climates, who are most liable to suffer from Ardent Fever.

SECTION IV. SYMPTOMS AND VARIETIES.

Simple Continued Fever presents different varieties, according to the circumstances under which it appears, and its duration.

[f] OZANAM, 1835, ii. 6.

The following is the clinical history of some of its chief varieties:—

1. The patient is seized rather suddenly with chills or actual rigors, followed by quick (100-120), full pulse; flushed face; dry, hot skin; white, furred tongue; great thirst and loss of appetite; confined bowels; scanty, high-coloured urine; severe headache, restlessness, and sleeplessness; or sometimes drowsiness, and pains or sensations as from bruising, in the limbs. These symptoms may subside suddenly, in twelve, twenty-four, or thirty-six hours, with copious perspiration, and then the disease resembles a single paroxysm of ague, and is appropriately designated *Ephemera.*

2. But occasionally, the fever is prolonged over several days. It may last four, seven, or even ten days (rarely longer), and it increases in severity with its duration. The pulse is 120 or more, and is full and often hard or bounding; the thirst and the heat of the skin are intense; the headache is more acute than in either typhus or enteric fever; sometimes it is described as throbbing or darting, and occasionally it is followed by delirium. The febrile symptoms usually terminate rather suddenly, with copious perspiration. This variety corresponds to the *Synocha* or *Inflammatory Fever* of many writers, and to the *Fièvre Synoque* of Davasse. The difference in duration alone distinguishes it from ephemera; the two diseases pass into one another by imperceptible gradations.

The crisis in ephemera and synocha does not always take place by perspiration. The cessation of the fever is sometimes attended by copious epistaxis, or hæmorrhage from the uterus or rectum, by vomiting or diarrhœa, or by a copious deposit of lithates in the urine. In a considerable number of cases, I have observed an eruption of herpes appear on the lips and face, towards the termination of the fever. This eruption, indeed, is so common, that some practitioners speak of *herpetic fever.* There is no characteristic eruption in any form of Simple Continued Fever. In a few cases, Davasse observed pale-bluish spots, not elevated above the surface, and not disappearing on pressure.[g] These are the *tâches bleuâtres* of French writers, already described and figured, which are occasionally met with in enteric fever, and in other diseases, and which, therefore, do not constitute a specific character (see page 474, and Plate V.).

3. The *Ardent Continued Fever* of the tropics is merely an exaggerated form of the synocha of Britain. The following is a brief account of the leading characters of this fever, as I observed

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[g] Davasse, 1847, p. 23.

it among the European troops at Calcutta in 1853, and in Burmah in 1854. The disease chiefly attacked the young plethoric recruits, recently arrived from Europe. It prevailed mostly in the hot, dry months (April and May), when the thermometer varied from 92° to 106° Fahr., and was never below 84°. In many cases, the symptoms commenced immediately after incautious exposure to the direct rays of the sun. The disease was ushered in with chilliness or rigors, or occasionally with nausea or vomiting. The pulse soon rose to from 100 to 120, and was full and firm. The other symptoms were: dry, burning skin; flushed face; giddiness; intense headache; ringing in the ears; intolerance of light; muscæ volitantes; restlessness and sleeplessness; tongue covered with a thick, yellowish fur; lips parched; great thirst; constipation, and scanty, dark urine of high specific gravity, depositing numerous crystals of lithic acid. About the fourth or fifth day, there was often acute delirium, followed by more or less unconsciousness, contracted pupils, and sometimes complete coma. Between the sixth and ninth day, death took place by coma, or there was a copious perspiration, followed by a rapid fall of the pulse, an increased flow of urine, an abundant deposit of lithates, and convalescence. The subsidence of the fever was sometimes followed by sudden, or even fatal, collapse.[h]

4. I am inclined to think that there is yet another variety of Simple Continued Fever, in which the symptoms are of a more asthenic character, and the duration sometimes more prolonged than in any of the varieties above-mentioned. The patient loses his appetite and strength; the pulse ranges from 90 to 120, and is rather feeble; the tongue is slightly furred; the bowels are confined; there is more or less headache, and the sleep is disturbed. These symptoms may continue for two or three weeks without any great change, except increasing weakness. On several occasions, I have known attacks of this sort follow great mental or bodily fatigue. This variety might be designated *Asthenic Simple Fever*. At the same time, it is to be remembered that pythogenic fever occasionally assumes characters, very like those now described (see pages 519 and 522).

Section V.—Complications.

When local complications occur in the course of simple continued fever, it is usual to regard the febrile symptoms as symptomatic of the local lesion. Cases, however, are occasionally met

[h] See Murchison, *On the Climate and Diseases of Burmah, Edin. Med. & Surg. Journ.* Jan. and April, 1855; also, Morehead, *Op. cit.* p. 165; and R. Martin. *Op. cit.* p. 204.

with, where the fever is out of all proportion to the local disease, or where the constitutional symptoms subside suddenly with free perspiration about the seventh day, and then it may be doubted if the local disease be not a complication of a primary fever. Cases of this sort, as Dr. Jenner observes, are apt to make medical men over-rate the efficacy of drugs in the treatment of acute diseases.

Section VI.—Diagnosis.

From what has been stated, it is obvious, that the diagnosis of simple continued fever is often impossible, until after its termination. It differs from typhus and pythogenic fever in its short duration, and in the absence of any eruption. An eruption of herpes on the face, about the fourth or fifth day of an attack of fever, would favour the supposition of simple fever, but is not incompatible with typhus (see page 133). The diagnosis may sometimes be assisted by the very severity of the febrile symptoms, which is usually greater at the commencement, than in either typhus or enteric fever. In this respect, simple fever more resembles the first paroxysm of relapsing fever, from which it is only distinguished when it occurs at times or places, in which relapsing fever does not prevail. The diagnosis between simple fever and acute local inflammations has been already considered.

Section VII.—Prognosis and Mortality.

Simple continued fever, when uncomplicated, is rarely fatal in this country. It may be doubted, however, if certain cases of fever, where death occurs under typhoid symptoms, and which are usually regarded as examples of typhus, without rash, be not examples of simple fever. The ardent fever of the tropics is a very serious, and often fatal disease.

Section VIII.—Anatomical Lesions.

Simple continued fever has no specific lesion. In the cases of ardent fever, which I dissected in India, there was great congestion of all the internal organs, particularly of the lungs, liver, and spleen. The right side of the heart was full of firmly coagulated blood. The sinuses of the brain and the pia mater were also very vascular, and occasionally there was an increased amount of intracranial fluid.

Section IX.—Treatment.

The simple continued fever of this country requires no special treatment. An emetic and a purge, followed by those measures

recommended under the head of typhus for relieving headache and procuring sleep, are usually all that is necessary. It is doubtful if diaphoretics hasten the natural termination of the disease; but there can be no objection to their employment. A mixture, containing small doses of nitre, with nitric acid and lemon juice, is much relished by the patient, and ought always to be prescribed, especially if there be any suspicion that the symptoms may be due to typhus.

For the ardent fever of the tropics, more active interference is necessary. All writers on Indian diseases recommend venesection, or leeches to the head, at the commencement of the attack, followed by the cold affusion, the continued application of cold to the shaven scalp, purgatives, and antimonial diaphoretics:[i] and from my own observations, I am inclined to think that life is often sacrificed by adopting less active measures.

For the asthenic form of simple continued fever, the best remedies are quinine and the mineral acids, with a nutritious diet, and a small allowance of port wine.

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[i] MOREHEAD, *Op. cit*, p. 166; MARTIN, *Op. cit.* p. 208.

CHAPTER VII.

On the Rate of Mortality of Continued Fevers at Different Places.

THE rate of mortality of each of the continued fevers has been considered in the preceding chapters. But in the official returns of most hospitals, no distinction is made between the different fevers; and therefore a few remarks are necessary, on the rate of mortality of the four fevers taken collectively. The following Table shows the rate of mortality of all the cases of 'continued fever' admitted into the London Fever Hospital during $14\frac{1}{2}$ years.

Table XLV.[j]

Years.	Total Fever Cases admitted.	No. of Deaths.	Rate of Mortality Per cent.
1848	1042	165	15·83
1849	401	65	16·21
1850	361	50	13·85
1851	614	43	7·00
1852	561	50	8·91
1853	787	149	18·93
1854	714	112	15·68
1855	622	113	18·16
1856	1300	230	17·69
1857	561	99	17·64
1858	239	36	15·06
1859	258	51	19·76
1860	151	37	24·5
1861	296	47	15·87
1862 to June 30	1239	245	19·77
Total	9146	1492	16·31
Deducting 97 fatal within 24 hours after admission	9049	1395	15·41
And 215 fatal within 48 hours	8931	1277	14·29

[j] The numbers given in this Table are made up of those contained in Table X, page 217, Table XXIV, page 366, Table XXXVII, page 529, and of the cases of Simple Fever, in Table XLIV, page 599. The numbers for 1848, include the 260 doubtful cases alluded to in note [f] page 51, of which 17 were fatal.

From this Table, it appears that out of 9146 cases, 1492 died, or the mortality was 16·31 per cent., or 1 in 6·11. Deducting the cases, which were almost moribund on admission, the mortality was 14·29 per cent., or 1 in 7.

The mortality from 'fever' in eleven other hospitals I have ascertained to be as follows:[k]—

	No. of Cases.	Deaths.	Mortality per cent.
St. Thomas's Hospital (1852-7)	1107	110	10·
St. George's Hospital (1851-6)	911	103	11·3
King's College Hospital, Dr. Todd's cases (1840-58)[l]	328	60	18·29
Newcastle Fever Hospital (1848-57)	1481	171	11·54
Nottingham General Hospital (1843-51)	845	108	12·78
Queen's Hospital, Birmingham (1852-7)	142	20	14·08
Royal Infirmary, Bristol (1840-57)	1890	179	9·47
" " Edinburgh (1840-57)	22586	2622	11·61
" " Glasgow (1840-53, & 1857-61).	22274	2575	11·56
" " Aberdeen (1840-61)	9191	852	9·27
Seraphim Hospital, Stockholm (1840-51)	3186	339	10·6

The excess of mortality in the London Fever Hospital is partly accounted for by the circumstance, that a large proportion of the patients are the aged and infirm inmates of the metropolitan workhouses. The remarkable effect of advanced age in increasing the mortality of typhus has already been pointed out (see page 221). The chief explanation, however, is to be found in the circumstance, that no fewer than 7546[m] of the 9146 cases (or 82·5 per cent.) admitted into the London Fever Hospital were examples of either typhus or pythogenic fever. Simple continued fever being rarely fatal, and the mortality of relapsing fever seldom exceeding 1 in 25 or 50, while the mortality of typhus is 1 in 5, and that of enteric fever almost as great, it is evident that the mortality from continued fevers, taken as a whole, must always rise in accordance with the preponderance of typhus and enteric fever. For example, in the year 1851, when one-half of the cases admitted into the London Fever Hospital were either relapsing fever or febricula, the total mortality from continued fevers did not exceed 7 per cent., or it was less than that in any of the hospitals above mentioned, over a series of years. Again, while the total mortality from 'fever' in the Glasgow Infirmary, is much below that of the

[k] These results have been obtained from the published reports, or from some of the officials, of the hospitals in question. The rate of mortality, for the individual years, will be found in a Table which I have elsewhere published (Murchison, 1858, No. 1, page 288).

[l] *Brit. and For. Med. Chir. Rev., Oct.*, 1860, p. 331.

[m] The 260 cases alluded to at page 51, note [t], are included in this number.

London Fever Hospital, that of typhus and enteric fever is about equal, and that of relapsing fever is more than twice as great (see pages 218, 367, and 529); the difference is mainly owing to the much larger proportion of relapsing cases included in the Glasgow returns (see pages 296 and 298). Lastly, in the year 1843, when the fever in Scotland was mainly relapsing, the total mortality from 'fever' in the Edinburgh Infirmary did not exceed 6·85 per cent., while in the Glasgow Infirmary it was only 4·5 per cent., and in the Aberdeen Infirmary 3·75 per cent. In fact, it is the large number of relapsing cases in this year, which makes the total Aberdeen mortality, as above given, less than that of the other hospitals with which it is compared.

It is obvious, then, that, in comparing the mortality from 'fever,' at different times and places, in order to judge of the merits of different plans of treatment, or for other purposes, it is absolutely necessary to take into account, not only the age and other circumstances of the patients, but also the species of fever which has prevailed. If this be not done, any such comparison is of little worth. It is also necessary, in drawing conclusions from the mortality of fever, to have recourse to large numbers. Twelve successive cases of typhus, terminating in recovery, may appear a remarkable success; but if 5 out of the next 8 cases die, the total mortality is 20 per cent. When large numbers are employed, there is found to be a striking equality in the rate of mortality of each of the continued fevers.

In Ireland, where fever has always been so prevalent, its mortality has been remarkably small, when compared with that of other places. This circumstance was pointed out in 1838, by Dr. Cowan, of Glasgow;[n] and was dwelt on at some length in my essay on the Etiology and Mortality of Fever.[o] Out of 156,809 cases admitted into the Dublin Fever Hospital, from the year 1817 to the end of 1861, I have ascertained that only 10,966 cases died, or the mortality was only 6·05 per cent., or 1 in $14\frac{1}{3}$.[p] In the Cork Fever Hospital, the mortality has been even less; out of 84,543 cases, admitted from 1817 to the end of 1861, only 3,354 died, or the mortality was only 3·96 per cent., or 1 in $25\frac{1}{5}$.[q] Moreover, the rate of mortality has varied much less in different years than it

[n] Cowan, 1838, p. 21. [o] Murchison, 1858, No. 1. Dr. Tweedie, in his Lectures on Fevers, published in the *Lancet* for 1860, has inadvertently transcribed, *verbatim*, my remarks on this subject, without, in this instance, indicating their source; consequently, a recent writer has been misled to quote, as from that author, a paragraph which appeared in my essay, in 1858.

[p] From data furnished by the Registrar of the Hospital.

[q] From data furnished by Dr. McEvers, of Cork.

has in England and Scotland. Thus, while in the hospitals of Britain, the mortality was in some years as low as 4 per cent., and in other years upwards of 20 per cent., in no year since 1817, has the mortality in the Dublin Fever Hospital reached 10 per cent., and at the Cork Fever Hospital, in only one year, has it exceeded 6 per cent.

The small mortality from continued fevers in the hospitals of Ireland is probably due to a greater preponderance of relapsing and of simple fever, than in the hospitals of Britain, for, as far as we know, typhus alone is as fatal in Ireland as in Britain (see page 218). It has been already shown, that relapsing fever is the principal fever which the Irish have at different times imported into Britain (see pages 56 and 300), while in mixed epidemics of typhus and relapsing fever, the proportion of relapsing cases has been much greater in Ireland, than in Scotland or England (see pages 39, 43, and 50). Dr. H. Kennedy remarks, that it is an erroneous impression, that the fever commonly present in Ireland is typhus. He adds:—'Under my own observation, I have occasionally seen 'the spotted cases (typhus) constitute one-third of those in hospi-'tal. In the epidemic of 1847-8, those with spots were very rare, 'not occurring oftener than once in fifteen cases. The disease, as 'seen then, was essentially a relapsing fever; at least, it was so in 'Dublin.'[r] Relapsing fever, however, is not a disease constantly prevalent in Ireland. From inquiries which I have made, I find that of late years, it has been almost as rare in that country as in Britain (see page 299). Hence, in the intervals of great epidemics, the small mortality of 'fever' in Ireland is probably due to a preponderance of simple continued fever, and not of relapsing fever. That this is really the case, appears from the report by Dr. McEvers, of the Cork Fever Hospital, for 1861, which is the only report of any Irish hospital, in which I have found the different continued fevers distinguished and classified.[s] From this, it appears that, during the year, 418 cases of 'continued 'fever' were admitted into the hospital, of which 254 were 'Syno-'chus or Simple Fever,' 116 'Typhus,' and 48 'Typhoid.' Of the 254 cases of simple fever, not one was fatal; of the remainder, 13 died.

It is a fit subject for inquiry, why cases of simple continued

[r] KENNEDY, 1860, p. 217.

[s] Whether the different Continued Fevers be regarded as species or varieties, it is much to be regretted, that the reports of most Irish and British hospitals afford little or no information concerning the number of each form annually admitted.

fever, which are the exception in the hospitals of England, are the rule in those of Ireland. There are some grounds for believing, that many cases of this fever in Ireland may be a modification of relapsing fever. In epidemics of relapsing fever, it has been noticed, that two of its most characteristic features—the relapse and the jaundice—have become less frequent as the epidemic declined, until the disease seemed to merge into what, under ordinary circumstances, would be regarded as simple continued fever (see pages 340 and 353). Dr. Steele, in his report of the epidemic at Glasgow in 1847, wrote thus:—'Towards the termination 'of 1847, and at the commencement of 1848, the relapse, usually 'considered the pathognomonic feature of the malady, was fre- 'quently absent, and the primary attack became prolonged to an 'indefinite extent; but the cases still retained the other symptoms 'characteristic of the disease, at this particular period. As the 'year advanced, and the numbers diminished, the relapses became 'less frequent, until they began to form the exception rather than 'the rule; and the disease ultimately assumed a mild form of 'synochus, the characters of which it has continued to retain to 'the present time.'[t] Dr. David Smith made a similar observation at Glasgow, in 1843.[u] Dr. Seaton Reid, in his report of the epidemic of 1847 at Belfast, distinguished between '*Relapsing* '*Synocha*' and '*Synocha*' or '*Febricula.*' Of the former, 1,014 cases came under his care, and of the latter, 1,238 cases, of which 23 were fatal. The latter had a duration or seven or eight days, and resembled the first paroxysm of relapsing fever, but was never followed by a relapse.[x] Lastly, Dr. Purefoy, writing of relapsing fever in Ireland, in 1853, observed:—'The disease yet continues 'in the country the same in essence, but modified by time and a 'variety of attendant circumstances.' Among the modifications, it is stated that the relapses were uncertain and irregular.[y]

[t] Steele, 1849. [u] Smith, 1844 (2). [x] *Irish Report*, 1848, viii. 303. [y] Purefoy, 1853.

CHAPTER VIII.

On the Relative Merits of Isolating Fever Patients or of Distributing them in the Wards of a General Hospital.

THE subject to be discussed in this chapter is one of great importance, on which considerable difference of opinion exists. On the one hand it is believed, that 'it would be better 'to have no hospitals at all, than to mix cases of typhus, small-'pox, and scarlet fever, with patients suffering from other 'diseases;' while, on the other, certain non-professional sanitary reformers, endowed with more zeal than knowledge, have proclaimed to the world that all cases of infectious disease ought to be distributed through the wards of general hospitals, and that Fever Hospitals, and Fever-wards are at all times 'a crime 'against humanity and a disgrace to the age in which we live.'

The establishment of Fever Hospitals in Britain dates from the commencement of the present century (see page 35). In 1802, Dr. John Clark of Newcastle collected the opinions of the most eminent physicians of the day, such as Dr. Matthew Baillie, Dr. Heberden, Dr. Saunders, Dr. Lettsom, Dr. Willan, Dr. Ferriar of Manchester, Dr. Haygarth of Chester, Dr. Falconer of Bath, Dr. Beddoes of Bristol, Drs. Gregory, Hamilton, and Rutherfurd of Edinburgh, etc., who were all strongly in favour of separate Fever Hospitals and Fever-wards, in preference to mixing fever patients with general cases.[z] For example, Dr. Ferriar observed: 'Previous to the establishment of fever-wards, when a patient 'happened to be seized with an infectious fever in the Manchester 'Infirmary, the disease was apt to spread to an alarming degree, 'so as to require a general dismission of the patients. But since 'these wards have been opened, though bad fevers have been 'accidentally introduced, yet by removing the patient on the first 'attack, the disease has always been prevented from extending.'[a]

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[z] Clark, 1802; see also Haygarth, 1801; and Stanger, 1802.
[a] *Med. Reflections*, vol. iii.

The circumstance, that most of the nurses and other officials of Fever Hospitals contracted fever, produced, after a lapse of years, a reaction in favour of the system of mixing the patients. In 1842, Dr. Graham of Edinburgh corresponded on the subject with many hospital physicians in London and elsewhere, including Dr. Bright, Dr. Williams, Dr. Latham, etc. Their opinion was unanimously hostile to express fever-wards, and favourable to the mixing of fever-patients with others; *provided the proportion was kept low.*[b] This opinion has since been advocated by Dr. Christison[c] and Dr. J. H. Bennett.[d]

In 1860, I issued, on behalf of the committee of the London Fever Hospital, a printed circular to 64 hospitals in the United Kingdom, with the object of ascertaining the present mode of dealing with fever patients. Replies were received from 40; viz., from 11 in London; 20 in the provinces of England; 4 in Scotland; and 5 in Ireland. Of the 11 London Hospitals, 8 admitted a very limited number of fever cases among the general patients, while 3, viz., University College, the London, and the Marylebone General Infirmary admitted no cases of fever. Of 20 hospitals in the provinces of England, 9 refused to admit fever-patients; 6 admitted them into separate wards, and only 5 distributed them among the general patients. There are also at least 6 hospitals for the special treatment of fever in the provinces of England. In every one of the 4 Scotch hospitals (Edinburgh, Glasgow, Aberdeen, and Dundee) there are separate fever-wards. In Edinburgh alone, the managers permit two fever-beds in each of the clinical wards (of 19 beds) for the purpose of instructing the students. Of the 5 Irish Hospitals, 1 was limited to fever cases; in 3 there were separate fever-wards; and in only 1 were the fever cases distributed among the general patients. Moreover, in most of the large towns of Ireland, there is a special hospital for the treatment of contagious diseases. With the exception of London, then, the prevalent custom is to isolate cases of contagious fever. The different practice in London is due partly to the desire of affording to students the opportunity of studying cases of fever, and partly to the circumstance that *a large proportion of the fever cases admitted into the London Hospitals are examples of enteric fever, which is never known to spread in the wards like typhus* (see page 430). But even in the London Hospitals, it is universally admitted that there is danger of true typhus spreading, if the number of cases is greater than 1 in 5, or 1

[b] GRAHAM, 1842. [c] CHRISTISON, 1850. [d] *Clinical Lect.* 2nd ed. p. 878.

in 6; so that practically, the necessity of a Fever Hospital for the surplus, which during epidemics may be enormous, is conceded. At the same time, it would not be difficult to adduce numberless instances where typhus has spread in a general hospital, when the above small proportion has not been exceeded, or where it has spread from even a single case. When I was a clinical clerk at Edinburgh in 1849, three typhus patients admitted into the clinical wards containing 38 beds, communicated the disease to 7 of the other patients, of whom 2 at least died. Dr. Stewart states that 'during the winter 'of 1837-38, an isolated case of typhus, in one of the medical wards 'of the Glasgow Infirmary, communicated the disease to most of 'those in the same ward, and several died.'[e] Dr. Peacock says, that when he was a student at Edinburgh, 'it was determined by 'the managers of the Infirmary, to try the effect of distributing 'the patients throughout the other wards, in the hope that the 'poison might by such means be so diluted, as to prove innocuous. 'As an experiment, four patients labouring under fever were 'placed in different parts of each ward, which usually contained '30 beds, and 4 beds were removed from the ward, so that each 'patient with fever, occupied the space of two ordinary patients, 'and their beds were placed in the piers between two windows, 'and these were kept constantly open. Notwithstanding these 'precautions, the fever spread to the patients in the adjoining 'beds, and in a month, no less than 12 such cases of infection 'occurred. Nor did the evil stop here; for one patient, who had 'been in a bed next to a fever patient, went out, sickened of fever 'after reaching her home, and spread the disease in a crowded 'court in Leith, previously free from any fever.' Dr. Peacock adds, that he has seen typhus spread in several other hospitals where the patients were mixed, although the space allotted to each patient was most ample.[f] Writing in 1838 of St. Bartholomew's Hospital, Dr. West states that out of 60 cases of typhus 14 had died, and he adds: 'Since last summer, 11 gentlemen, who were 'in the habit of frequenting the hospital, have been attacked by 'the fever, to which 3 have fallen victims; 16 nurses and 21 'patients admitted for other affections have likewise suffered from 'the disease, which terminated fatally in 10 instances, and I do 'not doubt that many similar cases occurred, which did not come 'under my notice. Seventeen of the 60 cases, to which my 'observations especially refer, were those of persons, who had 'either been in attendance on the sick, or who had at least lived

[e] STEWART, 1840, p. 297. [f] PEACOCK, 1856 (1), p. 162.

'in the same house: 9 of the 17 were the cases of patients who, 'admitted into the hospital for other affections, were while there 'attacked by fever. . . . It was at last found necessary from 'the disease spreading from bed to bed, to close one of the female 'wards.'[g] The spreading of the fever, under these circumstances, has been attributed to a want of due precautions; but I believe that it is impossible to make any rules, which, in practice, will prevent communication between the patients, on the mixed system.

The objections which are usually urged against Fever Hospitals and Fever-wards are two, viz.: 1, that the concentration of the poison increases the mortality among the patients themselves; and, 2, that the concentration of the poison increases the danger to the attendants. But these objections, which are too often made, without reflection on what would be the alternative if all cases of fever were admitted into general hospitals, apply only to fever-hospitals which are overcrowded or badly ventilated. *If* 2000 *cubic feet of space be allowed to each patient,*[h] *and if there be thorough ventilation, there need be no more concentration of the poison in a fever-hospital, than in a general hospital with a sprinkling of fever cases.*

Take, for example, the London Fever Hospital. It has already been shown that the rate of mortality *for each individual fever* has not been greater than what has been observed in most general hospitals (see pages 217, 366, and 529); while during 14 years (1848-61), 3680 cases of true typhus fever were treated within its walls; but the disease was only communicated to 53 persons, of whom 14 died. In other words, only 1 person caught the fever for every 70 under treatment, and only one died for every 263 under treatment. What would have been the consequences if these 3680 typhus patients had been distributed among the general hospitals of the metropolis, may be imagined from the comparison about to be made.

During the first six months of 1862, 1107 cases of true typhus were under treatment in the London Fever Hospital, of which 232 died, or the mortality was 20·95 per cent. In the same period, 343 cases of typhus were under treatment in 6 of the general hospitals in the metropolis, mentioned below, of which number 80 died, or 23·32 per cent. The 1080 (1107—27) cases admitted into the Fever Hospital communicated the disease to 27 persons, of whom 8 died. In other words, only 1 person took the fever for every 40 admitted, and only 1 died for

[g] WEST, 1838, p. 144. [h] This is the allowance at the London Fever Hospital.

every 135. But the 272 cases admitted into the six general hospitals communicated the disease to 71 persons, of whom 21 died; or 1 person caught the fever for every 3·8 cases admitted, and 1 life was lost for every 12·9 cases admitted. What would have been the result, if there had been no Fever Hospital, and if the 1080 cases admitted into it had been distributed among the general hospitals in addition to the few hundreds which were actually treated in them?[h] Yet, in the midst of this epidemic, the abolition of the Fever Hospital has been advocated!

Hospitals.[i]	No. of Admissions of Typhus.	Cases contracted in Hospital.	Total.	Deaths.
St. Mary's—Jan. 1st to June 30th, 1862 . .	16	1	17	3
St. Bartholomew's, „ „ „ . .	89	23	112	30
St. Thomas's, „ „ „ . .	92	12	104	16
Guy's, „ „ „ . .	40	21	61	21
Middlesex—Jan. 1st to Sept. 30th, „ . .	25	6	31	8
German—Dec. 1st, 1861, to Feb. 28th, 1862 .	10	8	18	2
Total	272	71	343	80

The question resolves itself into this. A certain number of cases of typhus having to be treated, how can this be done with most advantage to the patients, and with least danger to the attendants? From what has been stated, I think it is legitimate to infer, that on the plan of isolation, provided there be ample ventilation, they can be treated with equal advantage to themselves and with far less danger to the attendants, in proportion to the number of cases treated. That the attendants even in a well-ventilated Fever Hospital run a greater risk than the attendants in a general hospital, there can be no doubt; but this is due, not to a concentration of the poison, but to the foci of contagion being more numerous. A stranger, I believe, may walk along the centre of the wards in the Fever Hospital, with as little risk of taking typhus as in a general hospital with a sprinkling of typhus

[h] At Guy's, if not also at St. Bartholomew's, the admission of typhus cases was temporally suspended, on account of the disease spreading.

[i] For the St. Mary's return, I am indebted to Dr. Broadbent; for St. Bartholomew's, to Dr. Edwards; for St. Thomas's, to Dr. Hicks; for Guy's, to Dr. Steele; and for the German Hospital, to Dr. H. Weber. In all of these hospitals, the cases of typhus originated among the attendants and other patients in the wards, into which typhus cases were admitted. Of the 6 cases in Middlesex Hospital, only 1 originated in a ward in which were typhus cases; still, there had been no such thing as typhus appearing in the hospital, until after the admission of certain cases at the begining of 1862. At King's College Hospital, where the number of typhus cases admitted during the period in question, probably did not amount to 20, 3 persons, at least, caught the disease, who were in closest communication with the sick, and 2 of the 3 died. At the Westminster Hospital, three of the nurses contracted typhus during the period in question.

cases, but if he comes in close communication with each patient, the danger will be in proportion to the number of cases of true typhus.

The comparison above drawn between the effects of isolating or of mixing cases of typhus would tell far more in favour of isolation, if the plan of isolation had been carried out more completely at the Fever Hospital, than it has been. Of the 80 cases of typhus which have originated in the hospital during the last 14½ years, 28 have been patients recovering from enteric fever, scarlatina, or other acute diseases. Patients recovering from enteric fever or scarlatina, have so often contracted typhus, and patients recovering from typhus and enteric fever have so often caught scarlatina, that the scarlatina patients at the Fever Hospital are now kept separate from the others; and I believe that the same rule should be adopted with regard to typhus.

The above considerations justify, I think, the following conclusions:—

1. Cases of pythogenic fever may be distributed in the wards of a general hospital with impunity.

2. It is doubtful if cases of typhus ought ever to be admitted into a ward with other patients; even in no larger a proportion than 1 in 6, there is danger of the disease spreading.

3. Fever Hospitals are absolutely necessary in all large towns liable to epidemics of typhus, and they ought to be provided with the means of rapid extension, in the event of an epidemic breaking out.[k]

4. There is no evidence that in a well-ventilated Fever Hospital, the mortality from continued fevers is greater than in a general hospital. (See page 605).

5. In proportion to the number of cases of typhus treated, the danger of the disease spreading is much less in the plan of isolation than in that of mixing.

6. Cases of pythogenic fever, scarlatina, and typhus ought not to be mixed in a fever hospital.[l]

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[k] Temporary buildings of wood and iron, are particularly adapted for the treatment of typhus patients. (See page 251).

[l] Since the above was written, I have ascertained that during the three months July, August, and September, 1862, 393 cases of typhus were admitted into the London Fever Hospital, of whom 79, or 20 per cent., died. During this period, only 3 persons contracted typhus in the hospital, or 1 for every 131 patients admitted.

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